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THE LESIONS OF LATENT SYPHILIS*

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I first became interested in syphilis in 1893, when I went from the University of Michigan, where I had but little opportunity for the study of this disease, to Vienna with its wonderful pathologic material and an abundance of syphilitic material. At that time Kolisko was giving his popular courses in gross pathology illustrated with the material from the autopsies at the Allgemeines Krankenhaus. Well do I remember an incident that occurred one day while Kolisko was conducting an autopsy. As he took off the skull cap he remarked at once to me, standing by: "This man is a syphilitic." I looked for evidence of a meningeal gumma, but saw only focal thickenings of the meninges. From these and not from the presence of a gumma, Kolisko had made his diagnosis. This was to me new pathology, for I had been taught that gumma was the only infallible criterion for the diagnosis of syphilis, and I had never seen a case of syphilis diagnosed as such in the absence of gumma. As the autopsy proceeded, Kolisko called my attention to the increased "Derbheit," toughness, of the body tissues, which he affirmed to be another important gross pathological sign of syphilis. This, he explained, was due to the fibrosis produced in old syphilitics as the result of the latency of the disease. Many times since that day has Kolisko's observation of the fibrosis occurring in latent syphilis come to my mind, and I have wondered at his keen powers of observation and correct deduction. They were founded upon pure observation and logical thinking alone, for Kolisko had no other criteria by which to make a pathologic diagnosis of syphilis in the absence of the gumma, which was at that time the only histologic criterion considered absolute for syphilis. But Kolisko had noted the common association of fibrosis with gumma, and reasoned that it could also exist independently of the latter lesion. Years afterwards, after the discovery of the etiologic agent of syphilis, the *Spirocheta pallida*, I noted the occurrence of fibrosis as the chief end result of old and latent syphilitic infection. Virchow made a similar astute diagnosis of chronic interstitial myocarditis due to syphilis, in the absence of gumma, as early as 1858. Finding such a diffuse syphilis of the myocardium associated with gumma, he reasoned that it could occur without gumma as well. Several years after reporting my observations on the finding of spirochetes in such diffuse areas of myocarditis, I ran across Virchow's observations for the first time. It was a great satisfaction to me to have my findings thus confirmed by his earlier publication.

The lesion of latent syphilis repeats the essential pathology of the primary lesion, the hard chancre. It is predominantly vascular and perivascular, consisting essentially of lymphoid and plasma-cell infiltrations which are derived from the proliferation of the cells of the vessel wall and surrounding tissue. Numerous macrophages and cells of fibroblastic type also appear in the infiltration, derived particularly from the perivascular reticulo-endothelium. The lumen of the blood vessels is reduced to a small caliber through the cellular proliferation and through a peculiar hyaline swelling of the cells of the vessel wall itself. A chancre is, therefore, a plexus of vessels infiltrated with cells arising *in situ* through proliferation. The vessels of the chancre show on cross-section a characteristic concentric thickening and infiltration. All of these changes are found in the later lesions of syphilis, even to the smallest perivascular infiltrations.

Smears made of serum obtained from the hard chancre, fixed for two minutes in absolute alcohol, dried in the air and then stained according to the Warthin-Starry silver method, show the

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spirochetes in great numbers. This is a better method for the identification of the *Spirocheta pallida* than the dark field method, inasmuch as the morphological characters of the organism are better shown and more easily recognized. Even in the stage of the hard chancre, spirochetes are present in the blood stream; a general infection has taken place even before the chancre has developed.

Very little is known of the pathology of the secondary stage of syphilis. We have had, however, in the last several years, five fatal cases of arsphenamine poisoning in individuals treated in the stage of roseolar eruption, with chancre still present. In the livers of these patients numerous focal necroses were found, which at first were thought to be due to the arsenical poisoning. On staining the sections with the silver-agar method these necroses were found to contain colonies of spirochetes. The kidneys of these patients showed enormous numbers of spirochetes being excreted through the kidney tubules. In some sections the spirochetes are caught in the act of passing through the capillary wall into the intertubular tissues. In the blood vessels the organisms lie against the endothelium, more or less flattened out. After passing through the capillary wall the spirochete regains its spiral form and approaches the tubules, head-on towards the lumen. In the tubules the organisms are in part disintegrated in epithelial phagocytes, and in part go to pieces in the lumen. In the phagocytes the organisms gradually assume a horseshoe form, which contracts down to a single granule. In these granular forms we are becoming more and more interested, as the appearances indicate that there is a definite granular stage of the spirochete.

Another feature of the secondary stage is the early involvement of the meninges. As early as four to six weeks from the appearance of the chancre, perivascular infiltrations, of the same type as that of the primary lesion, although much smaller, may be found in the meninges containing spirochetes. Larger ones, suggesting miliary gummas, are rarely found. In clots made from the spinal fluid by the Alzheimer method spirochetes may be demonstrated in sections by the Warthin-Starry silver method. In a series of cases reported by Dr. Wile and myself some years ago, the demonstration of spirochetes in such clots by this method was successful in 12 out of 110 cases of syphilis examined.

Under the modern treatment of syphilis there are no secondary symptoms of syphilis. A period of ten to twenty-five years may elapse before symptoms of the latent infection become again manifest in the patient. In some cases the disease may never again become clinically active. But should such a patient die at any time an autopsy and thorough microscopic study will reveal the presence of small latent lesions, varying greatly in degree and extent. In other words, the modern treatment of the disease, like the old, results only in a clinical cure; in no case have I seen an absolutely inactive instance of syphilitic infection. On a careful search, active latent lesions have always been found in my material. It is possible that wholly inactive, cured cases of syphilis exist, but I have never seen such a case. Moreover, under the modern treatment of syphilis the period of latency seems to be shortened; neurosyphilis and aortitis appearing to be developing earlier (ten-fifteen years) than under the old treatment. On the other hand, the skin lesions, gummata and bone lesions seem to be decreasing in incidence. Numerous European syphilologists have recently expressed the opinion that the immunity conferred by the development of the secondary skin lesions is more lasting than that resulting from the early use of the arsenicals.

The use of the term, active latent syphilis, refers wholly to an infection not clinically evident, but which on microscopic examination shows perivascular infiltrations and proliferations. Such lesions are found usually in the aorta, myocardium, meninges, brain, liver, pancreas, adrenals and testes. Individuals with lesions of latent syphilis usually die of cardiovascular disease; a certain proportion develop central nervous system disease, cirrhosis of the liver, or pancreatitis with or without diabetes; while a still smaller proportion may show symptoms of adrenal insufficiency, or more rarely gastric or pulmonary syphilis.

Nearly 100 per cent (97.5 in 1910-1919; 83.2 in 1920-1930) of cases of latent syphilis show focal lesions in the meninges. There is apparently a slow progressive development of spirochete localizations in the meninges with healing and fibrosis. Nevertheless, microscopic examination frequently reveals the presence of minute areas of infiltration with lymphocytes and plasma cells. They are found most frequently over the parieto-frontal-temporal regions. They vary greatly in size; every degree of transition between mild fibroses to clinically active and more extensive involvement of the meninges may occur. It is easy to miss the milder cases on gross examination; often the sharply focalized thickenings can be seen only by oblique light or by floating the membrane upon water.

In the brain of the latent syphilitic, there will often be found on careful search small localized perivascular infiltrations, similar to those found in the brains of cases of paresis and cerebral syphilis. They are usually very few in number and are of very small size. They are found most frequently in the frontal and parietal lobes, and especially in the neighborhood of the ventricles. In many cases only one or two such infiltrations may be found on the most careful examination. All transition stages in the degree and number of these active infiltrations may be found, up to those of the more severe forms of brain syphilis and paresis. In cases of syphilitic psychosis these active lesions are more numerous than in the case of latent syphilis presenting no clinical evidence of cerebral disturbance, and are usually associated with localized atrophy and gliosis. Such lesions are found especially scattered throughout the frontal lobes. In these active perivascular infiltrations spirochetes of enormous size are often found. It may be stated as a fact that every case of latent syphilis has localized lesions resembling those of paresis, differing only in the extent and size of these lesions. As a rule the meningeal lesions occur more frequently and are more marked than the cerebral. Similar lesions are found less frequently in the meninges of the cord and in the substance of the cord than in the brain.

The hearts of practically all male latent syphilitics show latent lesions, varying from few and minute microscopic areas of increased stroma nuclei, plasma cells and lymphocyte infiltrations up to more diffuse areas of interstitial myocarditis. Gummatous lesions are rare. The majority of syphilitic women do not show such cardiac lesions, but nevertheless, some women may present as severe and diffuse forms of syphilitic myocarditis as are found in the male. The septum, anterior and posterior walls of the left ventricle near the apex are most frequently involved. In a few cases the right ventricular wall has shown more marked involvement than the left. The lesions are usually intermuscular rather than perivascular, representing perivascular increase of nuclei along the coronary capillaries. These infiltrations usually are not visible to the naked eye; in some severe cases they appear as irregular diffuse areas of pale translucency, without pigmented or hemorrhagic borders, becoming less translucent in proportion to the degree of fibrosis. In the healed stage of fibrosis the gross picture is that of the fibroid heart. At autopsy there is usually found dilatation of the left ventricle or of both. In the great majority of cases there is no valvular lesion; in a certain number of cases there is an associated aortic insuffiency. The writer has never seen a syphilitic lesion affecting the mitral valve. Coincident bacterial endocarditis may occur in some cases. In the examination of fifty hearts from asylum cases of paresis latent myocardial lesions were present in all. "Rheumatic heart" is the most common clinical diagnosis of the cardiologist for the myocarditis of latent syphilis and is made even when there is no evidence of valvular disease. Spirochetes are usually present in small numbers in the infiltrations. Often they are numerous.

The section of myocardium shown in the lantern slide is from a man who contracted syphilis at the age of eighteen. He was thoroughly treated according to the old mercurial method. At twenty-eight he developed optic atrophy, losing much of his vision. However, he had no more symptoms of the disease, and repeated Wassermanns in his later years were negative. His marriage was childless. At the age of fifty-eight he complained one morning of not feeling well and remained at home; after luncheon he went into the bathroom, where shortly afterwards he was found dead upon the toilet seat. At autopsy the heart showed to the naked eye a marked dilatation of the left ventricle with slight yellowish discolorations of the myocardium that were thought to be due to fatty changes. On microscopical examination they were found to consist of the extensive intermuscular infiltrations of plasma cells and lymphocytes seen in the picture. It seems incredible that a heart showing such extensive lesions should have given rise to no clinical disturbances until a few hours before his death.

The next picture is that of the myocardium in a young man under thirty years of age, admitted to the University Hospital, suffering from severe decompensation, without valvular lesion. The heart grossly showed the most extensive pale yellowish translucent areas throughout the wall of the left ventricle. Microscopically these all showed the marked cellular infiltrations characteristic of syphilis shown in the picture.

The youngest case of acquired syphilitic myocarditis is that recently reported by Dr. Wile and myself of a boy of twenty-four, with malignant syphilis. The entire myocardium showed diffuse syphilitic infiltrations with small gummatous areas.

Spirochetes are found in varying numbers in the myocardial lesions. In general, it may be stated that a cardiac death is the most frequent form of death for the latent male syphilitic, and that a very large proportion of the deaths that occur in middle life from so-called "myocardial degeneration" are in reality myocardial insufficiencies due to the changes resulting from latent syphilis.

Lesions of latent syphilis are found in the aorta of practically all cases of syphilis in the male, and in the majority of female cases, although much less marked in the latter. Gross examination of the aorta may not suggest the

presence of syphilis. The aorta may appear normal, or, as is usually the case, present more or less the appearance of atherosclerosis. Only infrequently are gross changes, in the form of thinning, porcelain-blue areas, irregular fissures, or longitudinal striae, characteristic of the gross appearances of aortic syphilis, present. Microscopically, however, the lesions of syphilis in the aorta show themselves in the form of perivascular infiltrations along the vasa vasorum. Aortic syphilis is essentially a disease of the vasa vasorum; the term mesaortitis is a misnomer. These perivascular lymphocyte and plasma cell infiltrations are always more marked in the adventitia of the aorta. There is found the same concentric thickening of the nutrient arteriole that is seen in the primary chancre. As the result of the obliteration of these vessels there is a progressive slow infarction, or fibrosis, of the intima and media. For the diagnosis of the occurrence of a syphilitic infection in any individual, the microscopical examination of the aorta alone would be most likely to reveal the presence of such infection. Spirochetes are sometimes present in the cellular infiltrations, but in our recent experience have been found most frequently in small areas of necrosis, infiltrated by polymorphonuclears. The paraaortic vessels apparently form the favorite seat for the localization of spirochetes in latent syphilis. Thickening of the walls of the arterioles occurs, with active plasma cell and lymphocyte infiltrations around the vessels.

The liver is frequently the seat of the lesions of latent syphilis in both sexes. They consist essentially of plasma cell and lymphocyte infiltrations of the periportal tissues, leading to fibrosis and to the picture of chronic hepatitis. The frequency of occurrence of atrophic cirrhosis and of hepar lobatum in latent syphilitics is very high. The latter condition in my experience has been met with more frequently in women than in men. Apparently hepatic lesions in latent syphilitics are on the increase; 15 successive autopsies on latent syphilis in my service a year ago showed hepatic involvement. Whether the modern treatment with the arsenicals bears any relationship to this apparent increase in hepatic lesions remains to be proved. An important observation is to the effect that women who have borne syphilitic children may remain symptomless otherwise until after the menopause, when they develop hepar lobatum. The demonstration of spirochetes in the hepatic lesions is less satisfactory than in those of any other organ.

In the pancreas, the lesions of latent syphilis are of the same perivascular nature. In the more severe form an interstitial pancreatitis may be produced, with fibrosis and loss of the islands, eventually pancreatic insufficiency and diabetes. I have been accused of saying that syphilis is the cause of diabetes. What I have actually said is that if interstitial pancreatitis is a cause of diabetes, then syphilis, as a frequent cause of pancreatitis, may become a frequent cause of diabetes. The majority of autopsy cases, on adult cases of diabetes, have been, in my experience, those showing interstitial pancreatitis associated with the lesions of latent syphilis. What part coincidence plays in this is not apparent.

The adrenals in the latent syphilitic show almost constantly small infiltrations of plasma cells and lymphocytes, varying in number and in size from a few small infiltrations scattered through cortex and medulla, up to diffuse infiltrations of marked degree, with interstitial fibrosis, parenchymatous atrophy and fibrosis of the capsule. These lesions are more frequent and severe in women than in men. The clinical picture in severe adrenal syphilis may be characteristically that of Addison's syndrome. In other cases, in which the clinical picture was not sufficiently marked to be diagnosed as Addison's, a fairly characteristic clinical syndrome may be present, which I have described as a "sub-Addisonian syndrome." The symptoms are characteristic; there are scattered pigmentations, low blood pressure, asthenia and a marked lumbar pain. In Brazil and Venezuela this syndrome has been recognized as due to adrenal syphilis, and cases of it have been reported as recovering under antisyphilitic treatment. Spirochetes can be demonstrated in the cellular infiltrations.

The testes of every latent syphilitic show sooner or later evidences of the presence of the infection in the development of a mild patchy interstitial orchitis, with atrophy of the germinal epithelium and a hyaline fibrosis of the basement membrane of the seminiferous tubules. The process is slow and prolonged over a number of years, but is progressive, and eventually the entire organ may become atrophic and hyaline. It may, however, preserve its normal size and consistency. In the gross, testes have been pronounced normal that microscopically showed no evidence of spermatogenesis, or were wholly fibroid. Latent syphilis produces a premature loss of spermatogenic function in the great majority of latent syphilitics. With this there is

frequently a premature loss of sexual power and desire, which may lead to suicide, as was true of several of our cases. In these cases the interstitial cells appear hyperplastic and contain a lipoid pigment. In only one case have I seen syphilitic lesions in the prostate, and that was in a young man aged nineteen, who had had his syphilis but two years, so that this cannot be regarded as a latent lesion. I have never seen a syphilitic lesion in the ovary or tubes that could be identified as such. In congenital syphilis spirochetes may be found in numbers in these organs, but without accompanying lesions. On the other hand, lesions with spirochetes have been found in the cervix, uterine wall and broad ligament.

In brief, such are the lesions of latent syphilis as shown by the ordinary autopsy material, representing the average middle and lower class population of a state like Michigan. In my series of autopsies in the University Hospital at Ann Arbor, the incidence of latent syphilis, as diagnosed on the microscopical criteria given above, was for the decade of 1910-1920, 43.7 per cent; for the decade 1920-1930 the average incidence was 25.7 per cent. In 1,675 autopsies lesions of latent syphilis were found in 408 males and in 86 females. The distribution of lesions in the various organs and tissues was as follows:

From a branch laboratory established in Honolulu, under Drs. Arnold, Fennel and Larsen, autopsy material has been collected and sent to Ann Arbor for microscopical study. Out of 140 cases examined, over twenty-five years of age, an incidence of active lesions was found in 70 or 48.6 per cent. As was to have been expected in a subtropical population, this incidence is higher than that found at Ann Arbor. Most authorities place the clinical incidence of syphilis as about 10 per cent in the whole population (Stokes); European authorities estimate it at somewhat higher percentages. The actual incidence cannot be known until autopsies are made on every one dying, and a thorough microscopic study shall have been made in every autopsy case. The ultimate diagnosis of syphilitic infection is the microscopic. As to the correlation between the Wassermann reaction and the pathologic findings, negative reactions are very common when the microscopic findings are positive; therefore a negative Wassermann has very little value in excluding the presence of latent syphilis. On the other hand, the Kahn test has shown in our experience a much greater degree of accuracy of correlation, particularly the so-called "presumptive Kahn test," which has shown very few errors in checking with the autopsy findings.

DISTRIBUTION OF LESIONS OF LATENT SYPHILIS IN 169 CASES OF SYPHILIS (142 MALES, 27 FEMALES) IN 386 AUTOPSIES OVER 25 YEARS OF AGE, 1909-1919. 43.7 PER CENT LATENT SYPHILITICS.

Tissue	Total Examined	No. Syphilitic	Per Ct. Syphilitic	No. Suspicious
Spinal cord	8	4	50.	
Brain	36	19	52.78	
Meninges	40	39	97.5	
Aorta	163	159 (6 aneurysms)	97.6	4
Heart	169	113 (3 aneurysms)	66.5	5
Liver	165	46 (4 gummas)	27.8	13
Adrenals	153	47	30.7	1
Pancreas	163	72	44.1	4
Testis	137	118	86.1	5

DISTRIBUTION OF LESIONS OF LATENT SYPHILIS IN 332 CASES OF SYPHILIS (275 MALES, 57 FEMALES) IN 1,289 AUTOPSIES OVER 25 YEARS OF AGE, 1919-1929. 25.7 PER CENT LATENT SYPHILITICS.

Tissue	Total Examined	No. Syphilitic	Per Ct. Syphilitic	No. Suspicious
Spinal cord	60	27	45.	1
Brain	198	71 (4 gummas)	39.9	2
Meninges	202	168 (2 gummas)	83.2	0
Aorta	327	282 (29 aneurysms)	86.3	7
Heart	330	216 (2 gummas)	65.5	7
Liver	328	102 (12 gummas)	31.4	4
Adrenals	320	145 (1 gumma)	45.3	2
Pancreas	328	98 (2 gummas)	29.8	0
Testis	264	203	76.5	10

(In this material were two gummas of lung, one of pulmonary artery, two of spleen, two of bone, one of thyroid, two of skin.)

DISCUSSION (*Abstract*)

Dr. Alfred Blumberg, Oteen, N. C.—At a former occasion you emphasized the existing pathologic difference of syphilis in the female as compared with that of the male. Would you give us some further information on the subject?

A Member.—What would your criteria be for distinguishing a syphilitic from a non-syphilitic focal mononuclear lesion?

Dr. Walter L. Bruetsch, Indianapolis, Ind.—Concerning the case of general paresis who lived many years I want to ask whether the paretic patient was suffering from any other incidental disease. Neuropsychiatrists are inclined to suspect in all cases of general paresis who live longer than five years some other complicating disease which counteracts the paretic brain process in a similar way as does therapeutic malaria.

Dr. Warthin.—As to the sex difference, the female shows not so frequently lesions in the meninges or in the brain of as marked a degree as the male. Yet we do find latent lesions in the brain in certain cases of psychosis that apparently are latent syphilitic. The aorta escapes; the heart escapes; but the liver and adrenals and pancreas show a higher degree of incidence in the female than in the male. I have examined thousands of ovaries, yet I have never found a lesion of syphilis in the ovary. Yet in the testis we find it, practically in every case of latent syphilis. There seems to be a marked difference biologically in the sexes in the localization of syphilis in the sex glands.

I often make a diagnosis of syphilis on histological changes alone now, because, after having found between 50 and 60 per cent of cases spirochete positive, I am sure that I can tell the lesions of syphilitic myocarditis from any other form of myocarditis. It is possible to differentiate rheumatic myocarditis and all other forms of myocarditis histologically. In recent epidemics of cerebrospinal meningitis that have occurred in Hawaii and also in Michigan there are present in the myocardium infiltrations that make one think of syphilis, yet there is an associated parenchymatous change that you do not get in syphilis and the infiltrations are not essentially plasma cells. I am quite sure there is a definite histological type of syphilitic myocarditis.

As to the man with paresis for twenty years, he had marked syphilis of the liver, syphilitic hepatitis, a very marked myocarditis, a very marked syphilis of the central nervous system, but no other disease, no history of malaria or other infection. It was a very extraordinary case in that he lived so long. There was nothing else to explain it; but other cases of paresis living as long have been reported. The cirrhosis in this case I regard as due to the syphilis.

Dr. Bruetsch.—We have observed at Indianapolis a case of general paresis who lived for sixteen years. Autopsy revealed besides the paretic brain process a cirrhosis of the liver which was held responsible for the prolonged duration of paresis. With the use of therapeutic malaria in the treatment of general paresis and other forms of syphilis slight degrees of malaria-cirrhosis of the liver are produced in a small percentage of patients who are given this treatment. Only recently we have observed at autopsy a well pronounced cirrhosis of the liver in a general paretic who a year prior to death had gone through a long course of therapeutic malaria.

Dr. Warthin.—Did he have a ruptured spleen?

Dr. Bruetsch.—No.

Dr. Warthin.—We have had three autopsy cases of paresis which had had malarial treatment, and every one had a ruptured spleen.

ENTERAL AND PARENTERAL FACTORS IN THE CAUSATION OF DIARRHEA*

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There has been a vast amount of speculation and scientific investigation concerning the causes, nature and treatment of infantile diarrhea. The fact that there is no characteristic pathology found in the gastrointestinal tracts of the majority of infants succumbing to this condition indicates that factors other than gastrointestinal damage must be operative, at least in those cases in which such specific diseases as dysentery can be excluded.

The fact that diarrheal diseases are more frequent in artificially fed infants, and that the feeding of certain types of food is especially likely to be followed by diarrhea, focuses attention on dietary factors. Each of the individual food elements, protein, fat and carbohydrate, has, in turn, been considered as the responsible agents. With increasing knowledge, however, it has been found that normal infants can safely be fed fairly large amounts of any of these constituents with impunity, provided conditions are otherwise favorable. It is true, of course, that the feeding of unusually concentrated foods may be followed by diarrhea or toxic manifestations, and also that in certain rare instances, as for example, in celiac disease, there is a specific intolerance for fat, but on the other hand, diarrhea of severe type may occur when minimal amounts of all of the food constituents have been fed. In these cases, some other explanation must be sought.

In some instances, diarrhea appears to be dependent upon constitutional factors, especially in the case of premature infants or in those known to be suffering from congenital abnormalities and perhaps in certain infants with especially sensitive and irritable gastrointestinal tracts or imbalance of the autonomic nervous

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system. None of the above mentioned factors, however, serves as an adequate explanation for the majority of cases of diarrhea. It has been a matter of common observation that infants fed on food which is subject to bacterial contamination are more likely to develop diarrhea than those whose food has been more nearly sterile, but except in the case of bacillary dysentery, paratyphoid and typhoid infections and certain other relatively rare types of bacterial food poisoning, it has not been possible to determine any specific bacterial cause of infantile diarrhea. In most cases of diarrhea, no abnormal types of bacteria have been isolated from the stools, and furthermore, diarrhea, even of severe type, may be observed in infants who have presumably received only food and water which was approximately sterile.

Diarrhea may occur suddenly in infants who have been receiving sterilized food of excellent composition and who have previously been thriving and have shown no symptoms of gastrointestinal disturbance. In such instances the onset of diarrhea is often coincident with the development of parenteral infections even in such parts of the body as remote from the gastrointestinal tract as the middle ears, respiratory system or genitourinary system. It would appear at first sight that the diarrhea resulting from parenteral infections must be essentially different from the diarrhea due to unsuitable composition of the food or to bacterial invasion of the gastrointestinal tract.

It is the purpose of this paper to point out the fact that diarrheas of enteral and those of parenteral origin are not essentially different in nature and that the treatment, except that directed to a correction of the parenteral disease, is, in general, the same.

In this discussion it is not proposed to consider those diarrheas which accompany specific disease, such as bacillary or amebic dysenteries, typhoid and paratyphoid infections or such digestive and metabolic disorders as celiac disease. We shall consider only that large group of diarrheal diseases usually designated as "simple diarrhea," "food diarrhea," "summer diarrhea," "fermentative diarrhea," "cholera infantum" or "alimentary intoxication." We include in this classification the diarrhea resulting from overfeeding, that accompanying parenteral infection and that due to bacterial contamination of the food by non-specific organisms.

There are certain features common to all of the diarrheas resulting from the causes mentioned. In all there is evidence of excessive bacterial activity in the upper portion of the gastrointestinal tract. Normally conditions are such in the stomach and upper portion of the small intestine that bacterial growth is inhibited even when fair numbers of organisms are introduced with the food, so that digestion and absorption proceeds uninterfered with by bacterial action and is fairly complete before the intestinal contents reach the colon.

When, for any reason, conditions are altered in the stomach and small intestine, so that bacterial growth is not inhibited, or when excessive numbers of bacteria are introduced by mouth or when the amount of food fed is in excess of the digestive and absorptive capacity, bacterial activity is vigorous in the stomach, duodenum and ileum. Numerous observers, notably Moro,[1] Plantenga,[2] Arnold,[3] Hartmann, Senn and Nachmani,[4] have found luxuriant bacterial flora present in the stomach and duodenal contents removed from infants in whom the above mentioned conditions have been present and have been associated with diarrhea. The bacterial flora of the stomach and duodenum in these instances is a varied one, but colon bacilli predominate and are the organisms most regularly found. These organisms may represent those which have been adventitiously introduced in the food and have survived, and this is obviously the condition existing when heavily contaminated food has been administered. In other cases, however, in which every precaution has been taken to avoid bacterial contamination of the food, there must be a different explanation.

There is very good evidence that microorganisms normally present only in the lower intestine may migrate upwards in the presence of favorable conditions, as for example, when an excess of food furnishing suitable bacterial pabulum remains unabsorbed until the colon is reached, thus converting the intestinal tract into a continuous culture tube. In such instances the withdrawal of the excess of food results in a discontinuity of the culture medium in the tube, because of relatively complete absorption before the lower portion of the small intestine is reached. With such a break in the continuity, the bacteria of the lower intestine do not pass beyond the gap and organisms which are harmless when in the colon, but potentially harmful when in the upper intestine, remain in their normal habitat.

One of the most favorable conditions for the growth of abnormal bacterial flora in the stomach and duodenum is a decrease in the secretion of gastric acid, which has a distinct inhibitory action on bacterial growth. When, for any rea-

son, gastric secretion is decreased, or when such acid as is secreted is neutralized by the food or by alkaline mucus, the stomach contents do not become sufficiently acid to inhibit bacterial growth, nor is acidity maintained in the duodenum. Under these conditions, and especially if the food is one which is readily fermentable, excessive bacterial action is an accompaniment of the diarrhea. Hartmann and Senn, working in our clinic, have demonstrated the close relationship between the degree of gastric and duodenal acidity and the extent of bacterial invasion. In infants with diarrhea of the types described, the presence of colon bacilli in the stomach contents is almost invariably associated with the greatly decreased acidity or hydrogenion concentration, and furthermore, when measures are adopted which result in increasing the acidity of the stomach, the bacterial flora quite regularly disappear and coincident with this there is generally a cessation or amelioration of the diarrhea.

Decreased gastric (and duodenal) acidity may result from a number of causes. Premature and undernourished infants, as a group, have gastric secretions which are notably deficient in acid. Excessive external temperatures lead to a decrease in the amount of acid secreted even by normal infants. Fever from whatever cause has the same effect. The giving of food of high buffer value, such as sweet cow's milk, not infrequently results in a fairly complete neutralization of such acid as is secreted into the stomach; the feeding of human milk, on the other hand, with its lower buffer value, is less likely to lead to a decrease in gastric acidity to a dangerous point. Any irritation of the pharynx or esophagus leads to an outpouring of alkaline mucus which is capable of neutralizing gastric acid. We see, therefore, that essentially the same favorable conditions for bacterial growth in the upper intestine may be brought about by feeding contaminated food, irritating food, food of a high buffer value, or an excess of easily fermentable food, or as the result of high external temperatures or of any infection resulting in fever, and furthermore that premature, very young, weak or undernourished infants are more likely to be affected by any of the factors mentioned because of the fact that their gastric secretions are at best slight.

Any infection, enteral or parenteral, which causes fever, predisposes to the development of an abnormal bacterial flora in the upper intestinal tract and consequently to diarrhea. There are, however, certain infections which are more likely to lead to this result than others giving rise to the same degree of temperature. Diarrhea is a much more frequent accompaniment and is likely to be more severe in cases of rhinopharyngitis and otitis media than of pyelitis or pneumonia. It cannot be stated with certainty just why this is so, but there are several possible explanations. In the presence of rhinopharyngeal infections, particularly those due to virulent strains of hemolytic streptococci, some of the organisms may actually be swallowed, survive in the gantrointestinal tract, and cause damage. The intense rhinopharyngeal irritation leads to a greater production of alkaline mucus, which is swallowed and mixed with the gastric secretion.

These explanations, however, do not hold in those cases in which diarrhea continues, and is severe, even after a throat infection has cleared and the only remaining demonstrable infection is in the middle ear or mastoid. When the infection is in these locations, the severity of the resultant diarrhea seems to be dependent upon the nature of the organism as well as upon the degree of temperature rise. Certain strains of hemolytic streptococci which are found in the presence of epidemics of rhinopharyngitis and otitis media are especially likely to be associated with severe watery diarrhea. In such an epidemic occurring a number of years ago, J. V. Cooke[5] studied the organisms isolated from our cases and found the streptococci to be of a toxin producing variety practically indistinguishable from the streptococci of scarlet fever. This, of course, suggests the possibility that the absorbed toxin may have a specific effect on the gastrointestinal tract. Middle ear and mastoid infections, due to staphylococci, even though associated with extensive necrosis and subperiosteal abscesses over the mastoid, are not usually accompanied by severe diarrhea. In the presence of pyelitis, even though the temperature may be high, vomiting is a more frequent symptom than diarrhea. In these cases there are often evidences of temporary pylorospasm which can best be explained on the basis of a reflex action. Such pylorospasm tends to prevent passage of organisms of the colon group into the stomach from below.

We have spoken of the type of diarrhea which is initiated as the result of parenteral infection, especially of the rhinopharynx, middle ear and mastoids, and have pointed out the fact that these infections act by bringing about changes in the gastrointestinal tract which are similar to those caused by other factors, such as food. There is another condition with which we frequently

have to deal in which parenteral infections are not the cause, but are the sequellae of diarrhea. Such cases are seen especially in undernourished infants who have developed diarrhea as the result of improper feeding or contaminated food. If the diarrhea persists for any length of time, the nutrition is necessarily further impaired and otitis media develops as a complication. The otitis in these cases usually comes on insidiously and is likely to be overlooked unless frequent examinations of the ear drums are made. Because of the fact that the infant's resistance is low, as the result of undernutrition, the middle ear infection is very likely to spread to the mastoid antrum and may fail to subside even after free and repeated paracenteses. In many of these cases the invading organism in the ear and mastoid is a colon bacillus or other intestinal type which may find its way to the ears from vomited material by way of the Eustachian tube or may be carried by the blood stream. The latter route is suggested by the frequent finding of positive blood cultures of *B.coli*. However the local infection is brought about, it is a complication which seriously prejudices the patient's chances of recovery by establishing what is essentially a vicious circle.

Having considered the nature and cause of the common diarrheas of infancy, let us turn to the treatment which should be based upon the known facts regarding the etiology. Whether the diarrhea be caused by overfeeding, bacterial contamination of the food or by such extraneous factors as heat or parenteral infection, treatment should in any event, be directed to correction of the abnormal conditions occurring in the gastro-intestinal tract. The food should be one which is within the limits of the digestive and absorptive capacity of the functionally impaired gastro-intestinal tract and an excess of readily fermentable food must, of course, be avoided. Furthermore, the food should be rendered free from harmful bacteria. Even of greater importance, means should be taken to correct the abnormal conditions present.

Of primary importance is the correction of the decreased gastric and duodenal acidity. This may best be accomplished by giving food which will not neutralize gastric acid and which, if possible, should be capable of maintaining the acidity of the stomach and duodenum despite the admixture of alkaline secretions. Some of the foods commonly used for the treatment of diarrhea fulfill these qualifications to a considerable degree as, for example, protein milk and the various types of lactic acid milk. Good as these types of food are, it is possible to improve them by the addition not only of acid but of "buffered" acid capable of maintaining its acidity. A suitable buffered solution is the one suggested by A. F. Hartmann, which consists of a mixture of lactic acid and sodium lactate. This solution is strongly acid and remains so even after the addition of considerable alkali. It can be mixed with milk or with dried protein milk and still maintain its acidity. The composition of the acid buffered solution is as follows:

BUFFERED LACTIC ACID SOLUTION

Lactic Acid U.S.P	15 c. c.
Sodium hydroxide, 10 per cent	20 c. c.
Water to	100 c. c.

Before use, the solution is diluted one part to ten with water.

In beginning treatment of diarrhea, the buffered solution alone is fed instead of water. A moderate amount of carbyohydrate, such as dextrimaltose or Karo syrup, may safely be added to the buffered solution, as the sugar is not likely to be acted upon by bacteria in the presence of such strong acid. Furthermore, a reasonable amount of added sugar (5-7 per cent) is absorbed from the intestinal tract about as rapidly as is the acid which protects it from bacterial action. After a few feedings of this type dried protein milk made up to the customary volume with the buffered solution instead of with water, or unsweetened evaporated milk mixed in the proportion of 2 volumes of milk to 3 of buffered solution, may safely be given and to either of these feedings a moderate amount of carbohydrate may be added. The feedings are continued until there is a decrease in the diarrhea or in the case of premature, very young or very weak infants, this type of feeding is continued indefinitely. No danger is to be anticipated from such a procedure. The buffered solution, although strongly acid, is metabolized in the body so as to leave an excess of base. Its use tends to correct any existing acidosis.

It has been found when the buffered solution or milk prepared from it is fed that coincident with the resulting increase in gastric and duodenal acidity, the abnormal bacterial flora disappears, and this is quite regularly associated with cessation or amelioration of the diarrhea.

Such treatment is equally effective whether the diarrhea be of enteral or parenteral origin, but if of parenteral origin, a restoration of normal conditions in the stomach and duodenum will be but temporary unless the acid feeding is continued or the parenteral infection receives

suitable treatment. Treatment of any parenteral infection is therefore indicated. In those cases in which the diarrhea is the direct result of abnormal conditions in the intestinal tract, brought about by a primary parenteral infection, excellent results are observed when the infection in question is eliminated. The treatment of the parenteral infections may involve local treatment of the nose and throat, paracentesis of the ear drum or in the case of epidemic streptococcus infections, with clinical evidence of mastoid involvement, post-auricular drainage or antrotomy. This latter procedure is rarely necessary except in epidemics. Such epidemics have occurred in widely separated portions of the country. We have ourselves not encountered such an epidemic for a number of years.

In those cases in which the middle ear or mastoid infections are secondary to diarrhea which has occurred as the result of other causes, local treatment of the infection serves only to remove a handicap which may seriously prejudice the infant's chances of recovery. The local infection is not the cause of the diarrhea, but when present tends to make the condition worse. Most patients of this type are in an undernourished condition and have but little natural resistance to infection. Operation on the ears and mastoids is not followed by the same brilliant results as in the case of the primary parenteral infections, for the original condition still remains. The wounds heal slowly and the mortality is high, in any event. A failure to recognize the existence of two entirely different types of association between parenteral infection and diarrhea has resulted in a considerable misunderstanding among pediatricians and otolaryngologists and is the cause of the divergent opinions which have been expressed as to the value of treatment of middle ear and mastoid infections as a means of controlling infantile diarrhea. Other infections, such as pyelitis, should likewise receive appropriate treatment.

Whatever the cause of the diarrhea, certain general measures are essential, such as maintaining the water balance and improving the nutrition and resistance by repeated transfusions. A detailed discussion of these procedures would, however, be beyond the limits of this paper. It should also be emphasized that the nutrition cannot be maintained unless adequate amounts of food are given and absorbed.

In conclusion it may be stated that no sharp distinction may be drawn between the diarrheas of enteral and those of parenteral origin; that whatever the cause, conditions in the gastrointestinal tract are likely to be the same and treatment should, in any event, be based upon the correction of these conditions.

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DISCUSSION (*Abstract*)

Dr. Philip F. Barbour, Louisville, Ky.—Those of us who have practised for any length of time have observed a great number of theories and suggestions as to infantile diarrheas. Each few years we are getting a rather different concept of what diarrhea is and what is causing it.

For many years the idea prevailed that diarrheas were only enteral and due to some type of bacterial infection in the bowel. We cannot question the fact that there are many cases of diarrhea that are definitely due to a type of infection in the food. Dr. Marriott has emphasized the fact that in many diarrheal cases there is no history of any change or any contamination of the food. A normal child fed on normal food should not have a diarrhea. In hospitals it is possible to say the child has had a normal clean diet, but when called into homes in private practice, especially into the poorer homes, it is difficult for us to say that the child has had a normal diet. Even if the milk was delivered as good milk we do not know whether it has been kept properly.

The Germans have vigorously advanced the idea that diarrhea is due to metabolic disturbances, that the child is unable to handle the food.

We are indebted to Dr. Marriott for insistence on the fact that diarrhea is sometimes an evidence of parenteral infection. Some of us rather went wild a few years ago, when operations upon the mastoid and upon the antrum were suggested to us, and were going to treat every case of diarrhea by an antrotomy. That was over-emphasized. The result, however, has been that the great majority of our profession have waked up to the fact that there can be infections in other parts of the body showing their effects in the bowel. It has made us all examine our cases more carefully for evidence of disease, wherever it may occur, and we are watching all parts of the child's body rather than focusing our attention solely on his gastrointestinal tract.

Dr. Marriott's suggestion that there may be an infection traveling up from the colon into the small intestine is valuable. I think he has gone very far toward solving the problem of treatment of diarrheas in our babies.

Dr. Robert A. Strong, New Orleans, La.—With the possible exception of infant feeding there is no phase of pediatrics that has been more perplexing to us than

the attempt to classify gastrointestinal disturbances. As Dr. Marriott has brought out, many of us have lived through various periods. We have studied the progress of these disturbances from the days of Beidert, tried to fathom the anatomico-pathologic conceptions of Weiderhofer, and have endeavored to discover the offending organisms described by Escherich in the bacteriologic era. We have fed "high fats" with Rotch and lowered them with Finkelstein, and we have suspected every food element as the cause of nutritional disturbances with Czerny and Keller. We have followed the acidosis studies of Howland and the present essayist or the teachings of Holt or Morse, and now we are beginning to appreciate more each day the role that parenteral infections play as an etiologic factor in gastrointestinal diseases.

When Dr. Marriott first announced this possible relationship, many of us were very skeptical, but I think his presentation today has clarified a great many of the misgivings we may have had concerning the theory which he really intends to convey. From all of the stages through which we have passed and experiences which we have had we must admit that the digestive capacity of the average infant is very wide, and we enjoy a great latitude in our feeding problems. Otherwise, I do not think that the babies would have survived the different foods which we have been feeding them from time to time.

There is another thing which Dr. Marriott brought out which should be emphasized: and that is that the normal reaction of the duodenum is now regarded as being acid, and that it is very important to maintain this acidity in the duodenum. This revelation, it should be remembered, completely revolutionizes our theory of the mechanism of the opening and closing of the pyloric valve as it was described by Dr. Cannon many years ago. As Dr. Marriott has shown, very definitely, the idea that the duodenum is normally alkaline is now believed to be erroneous.

There is no question in my mind that parenteral infections are the underlying cause of diarrheas much more frequently than we believe. In New Orleans, we have frequently seen cases in which the gastrointestinal disturbances were undoubtedly due to mastoid or other infections. There are other occasions, however, when this relation could not be established and I am glad that Dr. Marriott has made it plain that he does not believe that all diarrheas are due to parenteral infections. He has been criticized for his attitude, but after hearing his talk today, I feel perfectly satisfied that he has been very much misunderstood and that many of his critics are talking about intestinal conditions entirely different from those which Dr. Marriott describes

Dr. F. W. Schlutz, Chicago, Ill.—I think Dr. Marriott's paper this morning will quite thoroughly answer a good many of his critics. He has been charged with taking a very extreme position, which, as I recall it, he at the time very ably defended. I must say I must count myself among those colleagues to whom Dr. Barbour referred, who were rather staggered by his first suggestion. Longer contact with Dr. Marriott completely altered my opinion. He is so persuasive in his contentions that one can not resist him.

Some of the earlier theories advanced are in line with the newer investigations in this field, bacteriologically and otherwise; and I think that we can very readily and with complete ease of conscience accept the explanation Dr. Marriott gives of this type of diarrhea and (what is much more to the point and of interest to all of you, I am sure) base our attack and type of treatment upon the theory he advances and along the lines he suggests.

The acid milk, which was so thoroughly championed here in this country, has, as you all know, been a very valuable acquisition in the field of infant feeding. The importance of acidity and alkalinity in gastrointestinal physiology and the things, either enteral or parenteral, that alter them, are exceedingly valuable to remember and consider. I agree with Dr. Strong that our theories regarding alimentary disease and its causation have progressed from year to year. We know more about it and have better scientific ground for what we think and do today than ever before.

Dr. Fritz B. Talbot, Boston, Mass.—There is one point that I should like to emphasize, which is perhaps one of my hobbies, which I think is not emphasized enough in pediatrics and in the whole of medicine. When I first started to practise medicine, diarrhea was considered a disease; but in the conception of today it is really not a disease at all, but a symptom in the same sense as headache, constipation, vomiting, epilepsy and convulsions are symptoms. I think a great many of the misconceptions and many of the faults that we, as general practitioners, fall into come back to the mistaken idea that we are speaking of a disease entity and not a symptom. Of course, there are going to be changes in the conception of what is the cause of this symptom, because there are many causes; and we all know that some cases are due to infections of the mastoids; that some cases are due to infection of the intestinal canal by bacteria through the blood stream, as in dysentery and typhoid fever; and (at least I know, because every time during my first five years in practice that I got up to speak before a medical society I had diarrhea) it can be a nervous diarrhea. There are, therefore, many kinds. Different parts of the country may have different causes of diarrhea. I am quite sure that what is seen in New Orleans is different from what is seen in St. Louis, just as what is seen sometimes in Boston may be different from what is seen in other parts of the country. At this particular time of year we have been dealing with a diarrhea that has been associated with snuffles and colds in the head, which we call intestinal grippe.

Dr. Marriott (closing).—The ideas that I have expressed this morning about the etiology of diarrhea, let me say, are not original; I have simply put together things that have been known for a long while. The idea of the ascending infection from the colon upwards I believe originated with Moro, and the role of gastric acidity has been known for a long while. This is merely an attempt to link up the things we have known.

Diarrhea is a symptom; it may be a symptom of many different diseases. There is no need for any difference of opinion regarding this one phase of the subject of diarrhea, namely: the effect of parenteral infections. Parenteral infections can cause diarrhea. They do not cause all diarrhea, but do cause a certain amount. Further, diarrhea can, by failure of nutrition, cause parenteral infection. When there is a parenteral infection which is the underlying cause of diarrhea, an attempt to cure the diarrhea without proper attention to the parenteral infection is likely to be futile.

CERTAIN PROBLEMS IN THE MANAGEMENT OF THYROID DISEASES*

By S. L. LEDBETTER, JR., M.D., F.A.C.S.,
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The purpose of this paper is to discuss briefly certain problems encountered in the diagnosis and treatment of diseases of the thyroid gland. It is based on the observation of approximately five thousand cases, and a detailed study of the diagnostic questions and the end results of treatment in fifteen hundred cases which I have operated upon. Despite the knowledge gained from this experience, I am often baffled by problems of diagnosis, and I am not entirely satisfied with my views regarding certain phases of classification.

Ordinarily the diagnosis of toxic goiter (toxic adenoma and exophthalmic goiter) is easy, and it has been further simplified by the estimation of the basal metabolic rate. The mere presence of an enlarged thyroid in a nervous individual does not of itself mean that he or she has a toxic goiter. The nervousness may have no relationship to the goiter, or it may be due to hypothyroidism instead of hyperthyroidism. Frequently we see thin, nervous, irritable women whose thyroid glands are enlarged, but whose metabolic rates are subnormal. Sometimes the gland is enlarged, but is being destroyed by the growth of an adenoma. In such cases, after removal of the adenoma, the metabolic rate goes upward toward normal again. In passing, it might be said that operative work on patients with low metabolic rates should be limited entirely to the excision of adenomas. All of the original thyroid tissue should be permitted to remain.

In taking the patient's history, we should inquire particularly about the appetite, for it is rare in hyperthyroidism to find that the appetite has been poor. It is nearly always increased, and a history of poor appetite should always make us look further for some other explanation for the nervous phenomena. Failure to realize the importance of such small details, especially before the estimation of the metabolic rate became a routine procedure, is one of the reasons why many patients who were thought to have mild degrees of hyperthyroidism have been operated upon without improvement.

Tuberculosis associated with an enlarged gland is sometimes mistaken for exophthalmic goiter, although occasionally one finds the two together. The tuberculous patient with hyperthyroidism always does badly, and so does the diabetic. Joslin and Lahey report a fairly large number of diabetics with exophthalmic goiter, but I have had only one such case. I find diabetes associated with other surgical conditions much more frequently than with exophthalmic goiter. It is, however, highly probable that quite a number of my patients would have shown a lowered sugar tolerance. It should be emphasized once more, "The presence of an enlarged gland associated with nervousness does not always mean hyperthyroidism;" neither does the apparent absence of an enlarged gland, associated nervousness and tachycardia, mean that the patient has not a hyperthyroidism. The gland may be substernal, or it may be hyperactive without being enlarged.

CLASSIFICATION

I have found the following classification useful:

(a) Simple goiter
  (1) Adolescent colloid
  (2) Adult colloid
  (3) Adenoma
    (a) Fetal } solid or cystic
    (b) Single colloid } solid or cystic
    (c) Multiple colloid
    (d) Malignant
(b) Toxic
  (1) Primary hyperplasia, or exophthalmic goiter
  (2) Adenoma
  (3) Mixed, hyperplasia and adenoma
(c) Malignancies
  (1) Carcinoma
  (2) Sarcoma
(d) Inflammations
  (1) Acute thyroiditis
  (2) Chronic thyroiditis
    (a) Tuberculosis
    (b) Syphilis

There is no confusion or difference in the opinion of medical men interested in thyroid disease regarding the classification, except the relationship or differences between the so-called exophthalmic goiter and the so-called toxic adenoma. Some think that they are one and the same, with slightly varying pathologic states. Others think they are entirely separate and distinct diseases. From my own observations I would say that in many instances there seems to be quite an overlapping that would incline one to believe that they are one and the same disease; but again certain facts make me think they are different. Generally speaking, with the pure hyperplastic gland, the nervous symptoms predominate, with the pulse rapid but regular, except late in the disease; while in the adenoma

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group the cardiac symptoms and features predominate.

In adenoma one often finds the patient with a low metabolic rate due either to a pressure atrophy of the normal gland or to a destruction of the gland by the overgrowth of multiple adenomas. If these adenomas are excised, then the normal thyroid tissue often regains its function with the metabolism going up toward the normal. Now the toxic adenoma often shows the same pathologic picture as does the non-toxic adenoma, and the pathologist cannot always tell the difference between them. However, a patient with a primary hyperplastic gland always presents toxic symptoms, and one can very often tell by examination of the gland the stage of the disease, whether it is early or late, and whether or not the patient has been taking iodine.

One also never sees exophthalmic goiter with a normal gland, and again one never sees a purely hyperplastic gland in a normal individual. Such patients are always toxic, but with adenoma one sees normal hypo- and hyperthyroid states. One also often finds a mixed gland and in such a case the patient presents both pictures, the nervous phenomena plus the degenerative cardiac features.

The effect of iodine does not solve the problem. When first used it always helps the patient with the hyperplastic gland. In adenoma it sometimes helps and sometimes fails or even makes the condition worse.

PROBLEMS OF TREATMENT

In the light of our present understanding, the treatment of toxic goiter is surgical. Our greatest problem is the difficulty we still have in seeing these cases early, before permanent damage has been done, and before the disease is so well advanced as to make the operation a very definite risk. I will state, however, that we are getting these toxic goiters earlier than we formerly did, but we still see too many patients who are completely broken down, with marked and permanent damage to the body mechanism. In most of these cases there is a history of having taken iodine over a long period of time. It is in this type of case that we get our mortality from surgery, and it is this type of case that, though we may improve the condition of the patients, we can never restore them to normal.

Iodine is of inestimable value in the preparation of the patient for operation. It is of great value in taking the patient through an acute crisis of the disease, and we also find it of very great value in lessening the severity of the postoperative crises. I would, therefore, say that its use should be limited to the preparation of the patient for operation, to the overcoming of acute crises, to lessening of the severity of the postoperative course, and again it may be used postoperatively in small doses intermittently, say for one week in each month for several months after the operation, in an attempt to control the growth of the remaining portion of the gland. I might add, however, that iodine is of definite value, given carefully and intermittently, in the treatment of adolescent goiter.

The symmetrically slightly enlarged colloid goiter in a patient without symptoms and with a normal metabolism demands no treatment, either medical or surgical. However, the patient should be kept under observation, as this kind of a gland is apt to produce trouble in the future. To operate or to give iodine to patients with this type of gland may result in a disturbed physiologic state. There is only one exception to this rule. When the gland is large enough to produce pressure symptoms, or to be very unsightly, then an amount sufficient to relieve pressure or to smooth the neck should be removed.

The simple non-toxic adenoma should always be excised, particularly in patients over thirty years of age. Small, non-toxic adenomas in young girls may be left alone for a few years. The danger of the development of toxic adenoma and of malignancy is too great to allow an adenoma to remain indefnitely. It has been my experience that toxic symptoms are more apt to develop in patients with multiple adenomas, while malignancy is more apt to develop from the solitary adenoma.

The treatment of toxic goiter, as stated above, is surgical, with the administration of iodine for a short period only, as a part of the preoperative preparation. When one says surgical treatment, one does not mean simply removing part of the gland. One means everything from the beginning of the primary preliminary preoperative preparation on through to the completion of the convalescent period with an operation in between. A gentle, careful operation, without loss of blood, the removal of the proper amount of gland, the protection of the parathyroids and recurrent laryngeal nerves, the avoidance of tracheal compression, perfect asepsis and a smooth scar are absolutely necessary, but the operation is only one feature in the treatment. The failure to observe every detail will result in disaster, so the surgeon must exercise every fac-

tor of safety from the time the patient reaches the hospital until he is well out of danger. A thoughtless remark or a brusque reception by the hospital admitting officer may so upset the patient that she goes into an acute thyroid crisis. I have seen a prolonged history and physical examination throw a patient into delirium. One has no hesitancy in saying that no surgeon, no matter how great his technical skill, has any business treating exophthalmic goiter unless he is prepared to control and exercise every factor of safety. A low mortality and low morbidity are possible only where strict attention is given to every little detail. It is most necessary that you be able to estimate the physiologic status of your patient, both before and after operation. And this knowledge can only be acquired by the close and careful observation of a large number of patients.

You must develop the "feeling" that your patient is either safe or in danger. You cannot read it and you cannot be told. Almost all the fatalities that I have had were due to my inability to estimate accurately the seriousness of the disease.

PREOPERATIVE PREPARATION

This may be summed up as follows: rest in bed, allowing the patient to be up for one hour in the morning and again in the afternoon. Absolute rest in bed we have found tends to weaken the patient too much. Visitors should be limited to half an hour twice daily and the patient should not be bothered with household affairs or other responsibilities. If the patient is in the hospital, which is to be preferred, every effort should be made to secure quiet, rest and a happy frame of mind. The usual plan, when the home surroundings will permit, is to give the patient his or her first week's preparation at home, spending the remaining few days before operation in the hospital.

Frequent sponging and minor attentions tend to promote rest and quiet. Some mild sedative is given each evening and, when necessary, small doses of bromide two or three times a day. The patient is never awakened for a treatment or for the taking of the temperature. Lugol's solution, 10 or 15 drops in a full glass of water, is given three times a day: at 10:00 a. m., 3:00 p. m. and at bedtime. This accomplishes a great deal, provided the patient has not been taking it steadily. If he has, then it does no good.

Formerly I gave digitalis to all patients with severe exophthalmic goiter for several days before and after operation, watching the effect very carefully. Looking backward, I cannot recall any bad results from its use. Lately, at the Mayo Clinic it has been abandoned, Plummer stating that the mortality was less without it. Following his advice, I have discontinued its routine use, but still use it when I feel that the heart of that particular patient needs it. Certain hearts need digitalis whether the patient has exophthalmic goiter or not. In other words, exophthalmic goiter is not a contraindication to digitalis in a failing, fibrillating heart.

ANESTHESIA AND OPERATION

These two cannot be separated. The operation must be accomplished with the patient quiet. Whatever anesthesia sequence is used, if the patient goes on the table quietly, remains quietly, and returns quietly to his room, then it is perfect.

It has been my observation that local anesthesia is the method of choice, reinforced by some other basal or basic anesthesia, which may be in the form of a drug or light inhalation anesthesia or both. Lately we have been using the morphine, colonic ether, morphine, novocain sequence, with light nitrous oxide inhalation or oxygen rebreathing when necessary. We have found no objection to this method so far. Lately I have also been using sodium amytal with equally satisfactory results, giving it orally, 3 grains at bedtime, 6 grains two hours before the operation and then morphine about a half hour before operation. The patients reach the operating room very quiet and drowsy, but some of them become fretful when the least bit disturbed and do not take the local infiltration well, requiring a light inhalation of nitrous oxide. We have found that local anesthesia is more satisfactory following colonic ether than after the use of sodium amytal. However, the sodium amytal sequence is easier and more satisfactory afterwards, particularly on account of the postoperative nausea, there being much less nausea after sodium amytal.

Once in a while the patient is doing well but develops a thyroid storm the day before the operation. In such an instance operation should be postponed until after the acute crisis has subsided. However, if you should happen to wait too long it will probably happen again.

As for the operation itself, technically it should be simply a careful, gentle dissection, with perfect hemostasis, protecting the parathyroids, the recurrent nerves, the tracheal fascia and removing the proper amount of gland. The latter is purely a question of judgment from the close observation of end results. In exophthal-

mic goiter the tendency is to remove too little of the gland. In toxic adenoma one must be careful to leave sufficient normal gland. In the case of a mixed gland, one must remove all adenomata and part of the hyperplastic gland. It is better, of course, to remove too little than too much.

POSTOPERATIVE CARE

Ordinary postoperative care is sufficient with non-toxic cases. It is in the care of the toxic cases that we meet a real problem. Our routine for a number of years has been briefly as follows:

(a) The patient is returned to the room and placed in a comfortable semi-Fowler position with an ice bag over the heart.

(b) There is supervision by a competent nurse, with frequent sponging of the face and arms with cool water to promote quiet.

(c) Drugs to promote quiet are given, such as morphine, codein and pantopon, hypodermically reinforced by chloral hydrate *per rectum*.

(d) Continuous proctoclysis is used, adding to each can of the solution 1 dram of Lugol's solution and 40 grains of chloral hydrate, increasing or decreasing the dose as is necessary.

(e) Fluids are given by mouth and in serious cases glucose and saline infusions in large quantities.

(f) Lugol's solution *per rectum* is given as stated above, by mouth as soon as tolerated, and in desperate cases sodium iodide intravenously.

(g) Digitalis is given when necessary.

(h) No visitors are permitted until the postoperative reaction has subsided.

PROBLEMS OF SUBSEQUENT CARE

After leaving the hospital the patient is instructed as to diet, rest and the resumption of activity. These patients are not well. The disease is controlled, but damaged organs require rest to be restored. If the damage was great they will be improved, but a severely damaged heart muscle and nervous system will never be restored to normal, and it is for this reason that we should encourage early operation.

Occasionally a patient remains well for several years and then has a recurrence requiring a second operation, the remaining portion of the gland simply undergoing renewed activity. Occasionally we see what we call persistent hyperthyroidism: that is, though the patient may be temporarily and partially improved, the hyperthyroidism still persists. This may be due to removing too little of the gland, or to lack of proper postoperative care, or may be due to the fact that the remaining portion of the gland continues to grow and to over-function. These cases will require a secondary operation, sometimes a third and a fourth, as I have once seen.

END RESULTS

Persistent hyperthyroidism, exophthalmic goiter	4
Recurrent hyperthyroidism, exophthalmic goiter	2
Recurrent hyperthyroidism, toxic adenoma	1
Persistent hyperthyroidism, toxic adenoma	0
Recurrent multiple adenoma, non-toxic	1

In this last case, at first operation the amount removed filled a quart fruit jar. Three years later the gland was as large as before. The patient also then had myxedema and acromegaly.

Postoperative tetany, temporary	1
Postoperative tetany, permanent	0
Injury to recurrent laryngeal nerve	1

POSTOPERATIVE HYPOTHYROIDISM

Exophthalmic goiter	1
Well seven years, then myxedema. Controlled with thyroid extract.	
Toxic adenoma	0
Multiple adenoma, non-toxic	1
Adenomas also recurred, requiring second operation.	
Simple adenoma	0
Colloid goiter	0

MORTALITY

Exophthalmic Goiter

After Thyroidectomy

Postoperative hyperthyroidism	2
Delayed death	1
Death on operating table	1
Total	4

After Ligation

Postoperative hyperthyroidism	1
Delayed death, a disease not controlled	3
Total	4

TOTAL EXOPHTHALMIC GOITER DEATHS

Postoperative hyperthyroidism	3
Delayed deaths	4
Death on operating table	1
Total exophthalmic goiters	8

TOXIC ADENOMA

Postoperative hyperthyroidism	0
Delayed postoperative death, cardiac (one week)	1

NON-TOXIC ADENOMA

Postoperative death, cause undetermined	1
Anesthesia death on table	1
Total deaths	11
Percentage mortality	0.73 per cent
Percentage excluding delayed ligation deaths	0.53 per cent
Deaths without operation	3 cases

DISCUSSION (*Abstract*)

Dr. W. O. Johnson, Louisville, Ky.—The presence of a goiter does not mean that the patient has a toxic goiter, even if he is nervous and has a slightly elevated metabolic rate, and some of the severest cases of hyperthyroidism occur in individuals who have no appreciable enlargement of the thyroid gland. So the absence of thyroid gland enlargement does not rule out the possibility of hyperthyroidism.

In addition to the important symptoms which Dr. Ledbetter has mentioned as aids in the diagnosis of hyperthyroidism are: persistently good appetite with rare gastrointestinal disturbances and progressive loss in weight. This triad is of great importance, also optimism, inattention which is interpreted by the individual as loss of memory, dilatorious effect of hot weather, and impairment of insight as to the seriousness of their condition.

Another form of iodine therapy in thyroid states is the prophylactic treatment of pregnant and lactating mothers, who have goiter, or are in goiter belts. This aids in the prevention of subsequent enlargement of the thyroid of the mother and development of fetal adenoma in the child.

I cannot stress too emphatically the importance of early diagnosis and operation before general systemic damage has occurred, and I also heartily agree with the essayist as to the need of removal of isolated adenomas before the patient is thirty, as a means of prevention of toxic adenoma or of malignant degeneration.

The use of common sense in the administration of digitalis is of paramount importance. The undamaged heart does not need digitalis, but the heart that is damaged, or has had previous cardiac damage, should be digitalized, like any other heart, regardless of the hyperthyroidism.

Dr. Ledbetter's operative set-up is widely used by those doing thyroid surgery, and I wish to stress the point that thyroid surgery includes a careful, detailed medical and surgical supervision from the time of diagnosis and preparation for operation, until restoration to health.

The one procedure which in my hands has been of greatest help in the handling of severe toxic cases or those with cardiac damage, a procedure which gives the most relief from worry, anxiety and fear, and which is not dependent upon special types of anesthesia is: to operate in the patient's room, in bed, or take the operation to the sick patient, and not make the sick patient go to the operation.

By doing this, when the patient is properly prepared, ligations and lobectomies are practically eliminated, complete operations can be done at one sitting, without added risk, with less reaction, a quicker recovery, and therefore, better end results.

Dr. John R. Wathen, Louisville, Ky.—We should draw a fine distinction in treatment and diagnosis between exophthalmic goiter and toxic adenoma. These are not difficult to distinguish in 90 per cent of cases.

The exophthalmic goiter is essentially a toxic goiter, and expends its energy on the central nervous system. The toxic adenoma is just the opposite. It expends its energy upon the cardiorenal system. The latter kind, of course, is the more dangerous type.

In exophthalmic goiter, as a rule, only one lobe is involved, sometimes both. The right lobe is usually much larger. In toxic adenoma the lobes are usually smaller. There is a difference in the preparation of the patient. Due to the work of Dr. Plummer, we have appreciated Lugol's solution in exophthalmic goiter. In the toxic adenoma, there is a question whether it is of value at all.

Dr. Plummer says that the mortality has been increased rather than decreased by administration of digitalis. Yet, cardiologists differ with him. My own personal experience has led me to believe that the cardiologists are right. If you operate upon a case, under local anesthesia, and about a week afterwards a crisis occurs, what are you going to do if you don't use digitalis? If you will inject one very large dose of digifoline into the vein you can break up the crisis almost immediately.

In the preparation of the patient, I give Lugol's solution entirely by rectum. Don't give 10 or 15 drops after meals. I use 100 drops once daily in a pint of water, at 6 in the morning by the Murphy drip, running rather fast. I get a large amount of iodine in rapidly and the benefit is considerable with cases of exophthalmic goiter. The patients with toxic adenoma have dilated hearts, and we have to be very careful in their preoperative preparation. The operation should be done exclusively under local anesthesia.

I have discarded intravenous sodium amytal as it puts the patients to sleep, and we cannot observe injury to the recurrent laryngeal nerves.

We are making progress today in distinguishing between these types of goiter, in determining the proper therapy and the type of operation.

Dr. Robert K. Buford, Charleston, W. Va.—The most confusing, complex, and difficult phase of the goiter problem, to the general practitioner, has been classification. Almost every man giving special attention to goiter has a classification of his own. No classification at present meets the ideal requirement of the clinician, the surgeon and the pathologist. The classification of pathological changes in the thyroid gland in the absence of any known etiological factor must be strictly referable to the physiological and anatomical bases manifest in the normal gland, and should not be obscured by the introduction of any clinical or theoretical consideration.

I have come to feel that the different types are related. I believe pathological evidence is tending to bear this out, and that it will ultimately be found that the differences are truly in degrees. Basing our conception of thyroid diseases, therefore, on the physiology, chemistry and pathology of the gland, we have come to the following clinical classifications:

(1) Colloid goiter
(2) Adenoma, or nodular goiter
(3) Hyperthyroidism
  (a) Graves' disease, primary.
  (b) Adenoma or nodular, secondary.

Pemberton has pointed out that except as to the fundamental principle involved, there can be no standardization of the preoperative preparation. Among patients with hyperthyroidism, the method must be adapted to the individual.

Iodine, rest and a high caloric diet are necessary for all patients. I find rest in bed in the hospital essential for the preoperative care of hyperthyroidism and by such treatment the condition of the patient is greatly improved. Hospitalization may be necessary from a few days to several weeks. During this period, heart and kidney function and the nervous stability of the patient are carefully evaluated. The result of this observation, combined with study of the basal metabolic rate, gives us a fairly accurate index of the degree of intoxication.

The most common complications which may result from thyroid surgery are injury to the recurrent laryngeal nerve, hemorrhage, acute hyperthyroidism and cardiac failure.

A thorough knowledge of the anatomy of the neck, and the performance of an anatomical operation, leaving a small portion of each lobe, will prevent injury to the recurrent laryngeal nerve and parathyroid glands.

Hemorrhage is controlled by ligating each vessel separately, avoiding mass suture, and the elimination of drainage whenever possible.

Cardiac failure should be prevented by selection of cases and preoperative care. Having decided that the case of the patient with thyroid disease is a surgical one, our problem is to give the treatment that will take the patient through the surgical experience with the least risks. Our preoperative treatment is divided into two classifications:

(1) Cases with compensated heart.

(2) Cases with decompensated heart.

Auricular fibrillation is not alone a sign of decompensation. A heart may fibrillate and still be compensated. We give digitalis only to cases with decompensation. We prefer Eggleston's method of dosage. When all signs of complete decompensation have disappeared, usually seven to ten days after complete digitalization, surgical treatment should be instituted. Quinidin sulphate is often invaluable in controlling the fibrillation in cases where no decompensation is present or after the heart has become compensated. Digitalis will not control tachycardia. Lugol's solution is far better.

We have always felt that the same amount of thyroid tissue should be removed in all types of goiter. The end results of 507 cases followed from one to ten years having repeated clinical and metabolic examinations, have fully confirmed my early expressions. The best results are secured when enough thyroid tissue is removed to produce a temporary hypothyroidism. The remaining portion of thyroid tissue should be kept saturated with iodine for about two months after operation and all foci of infection should be removed to prevent regeneration of thyroid tissue and recurrence of the disease.

We believe with Smith, Crute and Strider that the incidence of myxedema in certain cases is due to the administration of iodine after operation to prevent hyperplasia and that a histological study of the thyroid tissue removed shows little apparent correlation between the degree of involution and the basal metabolic rate and clinical pictures. The preoperative administration of iodine is one of the chief factors in disturbing this relationship.

I should like to warn against using iodine routinely after operation if the patient gives a history of a previous thyroiditis. These patients are potential candidates for myxedema and should never receive iodine after operation.

I want to call your attention to three factors in hyperthyroidism: (1) the stimuli; (2) the changes in the thyroid gland and its resulting disturbed function; and (3) the effects of disturbed secretion upon the cells of the body. The cells that are most commonly affected are those of the heart muscle. The heart changes are the only ones that we can study with any degree of scientific accuracy. The basis of sound surgical treatment of hyperthyroidism is early diagnosis, preoperative management, determining the operability, selecting the optimum time and the type of operation.

Surgery offers more in the treatment of goiter than any other therapeutic measure, in an institution where the internist, surgeon and pathologist have equal opportunity for the study of the disease and where the team work is perfect. I know of no results in surgery which are more immediate, more lasting and more satisfactory than those following the proper surgical treatment of goiter.

Dr. M. Casper, Louisville, Ky.—I think the procedure of all goiter surgeons is to remove as much of the thyroid gland as possible. We have all seen, in cases which we left a little nodule at the superior thyroid pole, a subsequent activitation in this nodule requiring a secondary operation for its removal. This is more prone to occur in an exophthalmic goiter than in any other type.

Some patients with damaged hearts are discharged much too soon. The damaged hearts should be treated over a long period of time by a competent man. They should have a graduated amount of exercise and systematic care. I have never been able to get digitalization in any toxic thyroid condition. I have tried all forms of digitalis, including Upshur Smith's, and have yet to find a single case in which we can obtain its physiological action.

These cases must be handled with a great deal of tact. Nurses who do a great deal of this work become very proficient in handling this peculiar type of patient. It is our routine to have specialized nurses at the hospital before the patient gets there; in fact, to have the nurse meet the patient at the door and have charge of him throughout the hospitalization.

Dr. Ledbetter (closing).—It should be emphasized that the subsequent care following operation for thyroid disease is of the utmost importance in the final good results that one strives to obtain. In the discussion it was mentioned that the indiscriminate use of iodine following operation may be a factor in the development of postoperative hypothyroidism. I am of the same feeling and if iodine is used during the convalescent period the patient should be under close observation with frequent estimations of the basal metabolic rate. The careful follow-up cases after thyroidectomy should be stressed. Frequent personal observations, say at least once a month for the first three months, and then every six months or oftener if necessary for a period of three years or longer, are indicated in the meticulous follow-up of these individuals.

GANGRENE AND NECROSIS OF THE UTERUS*

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From the scarcity of literature on this subject, especially in America, it would seem that the above mentioned conditions are extremely rare. However, in the gynecological service of Baylor Hospital several cases of gangrene involving the uterus have been observed, two of which will serve as the basis of this paper.

The uterus, of course, undergoes decided changes during the pregnant stage. Generally speaking, gangrene is most frequently encountered in tissue which has a lowered resisting power. In some instances it seems that the more highly organized tissues of the body offer less resistance to the causes of gangrene, as it is frequently found where a very abundant circulation exists. A distinction probably should be made between necrosis and gangrene. Necrosis is generally defined as the death and degeneration of single cells or larger groups of cells with concurrent changes in the intercellular elements. A slight degree of necrosis may escape detection by the ordinary means of diagnosis and may be revealed only by microscopic examination. Gangrene is known as that condition of the tissues of the body in which we find necrosis combined with putrefactive changes. Primary gangrene is produced by specific gangrene-producing bacteria, or the ordinary bacteria acting on already devitalized tissue. Moist gangrene usually occurs in tissue or organs ordinarily exposed to infection by saprophytic microorganisms.

Before going into the causes of the subject of this paper let us consider some of the factors connected with the circulation of the blood.

The first and most important one is the arrest or disturbance of circulation. Any marked interference with normal circulation in a part will bring about a degenerative process. If the tissue is highly resistant, a temporary interference with circulation may not cause extensive death to tissue, and it is possible that almost complete restoration of function may occur. But certainly, if the circulation is interfered with for any length of time, necrosis, or the more definite form of tissue death, gangrene, will result. The rapidity with which collateral circulation is established in an area or an organ determines the possibility of a return to normal in that tissue after it has been affected by diminished nutrition.

A slide showing the injected blood vessels of the uterus was taken from Norris' work, but I think was originally from some other source. This slide very graphically shows the marked anastomosis of the blood vessels and the very great number of blood vessels supplying the uterus. After studying this picture one would think that it would be almost impossible for the blood supply of the uterus to be so interfered with as to produce gangrene. In this connection I have found it interesting to take up some study of the physiological function of the capillaries in general. At the risk of making my paper too long, allow me to refer to the work of Krogh, of Copenhagen University, in his Yale-Silman lecture course. His studies embraced the capillaries in almost all structures of the body, but no definite reference is made in them to capillary circulation of the pelvic organs. There is probably no marked difference in the capillaries of the pelvic organs and those of many other parts of the body. Their number is astounding and has been found to be fairly constant in muscle tissue; for instance, a space the size of the transverse section of an ordinary pin can carry 700 parallel capillary tubes and 200 muscle fibres. The total length of capillaries in an average individual has been estimated to be 100,000 kilometers, or 2.5 times the distance around the globe.

It is essential to the life of muscle that it have oxygen and water. Other substances, of course, are given to the tissue, but apparently the most necessary substance is oxygen, supplied largely by the capillaries. This and other reasons seem to establish the capillaries as one of the most essential parts of the circulation. It was generally thought that the usual pressure and circulation within the capillaries was due to pressure in the arterioles. However, it is very evident that there is another force entirely independent of the arteriolar pressure, which causes relaxation and contraction of the capillaries, and this is probably the factor which allows capillaries to supply the muscles under the minimum as well as the maximum demand. After the fact of independent capillary contractility was made known, the question to be determined was, by what means can contractions be carried out?

Hooker, in 1920, observed that the outside diameter of a capillary did not become appre-

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ciably altered, even when the lumen was diminished, so it was thought that the decrease in internal diameter was brought about by swelling of protoplasm and imbibition processes. The next view offered suggested the existence of contractile cells in the capillary wall. It was thought that oblong nuclei were discovered, arranged in the direction of the tube, surrounded by protoplasm, which in many places formed hoops or loops. These structures were thought to be related to the smooth cells found in larger vessels. It was concluded that the fibrils on the branched cells on the arterioles represented the contractile elements in the cells, which influence the whole capillary contraction. The contraction begins in these cells. These richly ramified muscle cells on the capillary walls, now well studied by Vimtrup, are the same as those described by Rouget, so are designated as Rouget cells.

Capillary tonus is subject to nervous, hormonal and other influences, which cause contraction and change in the diameter of the vessel. The new conception of capillary contraction, developed by many investigators, and exhaustive work by Dr. Krogh, is that the calibre of vessels is mainly determined by their own tonus. They, also, of course, are influenced by the well known pressure of blood upon these walls. The contractile coat of the capillaries may be influenced by various stimuli, for example: through the nerves, temperature, and light. Chemical messengers through the blood, the hormones, pituitary hormone, suprarenalin, histamine, also have a direct influence upon the capillaries.

Following this consideration of the physiology of the capillaries, perhaps something of a practical nature can be developed bearing on the subject of necrosis and gangrene. As long as the capillary tube and current are normal, no cessation of circulation is noted.

When the capillary blood pressure is higher than the osmotic pressure, an excess of water will filter through the capillary wall and edema will develop.

The passage of red corpuscles through the capillaries is quite interesting, as often the capillary is so small that only a single file of corpuscles may pass; while other vessels are so narrow that the corpuscle can pass only after being deformed for a moment.

True stasis, observed in the capillaries, due to packing of corpuscles and absence of plasma, usually is broken up by pulsation and presence of fluid, the corpuscles being forced beyond the stagnant area into the blood current.

Diapedesis, in inflamed tissue and from capillaries which have gone into stasis was carefully studied as far back as in Cohnheim's time, in 1877. Diapedesis may occur in this manner without the wall of the vessel suffering permanent injury. Circulation and emigration of white corpuscles is quite like that of red cells. When the demand for emigration comes, the leucocytes are strongly drawn to or beyond the vessel wall and many capillaries may be blocked, while the leucocytes find their way into the tissue. This penetration damages the vessel but little as the openings close quickly, unless damage to the tissue has occurred.

Means has said that however obscure the functions of the endocrine glands may be, they are all concerned in one way or another with metabolism, and likewise with the circulation.

"If the secretions of the endocrine glands influence cell activities, they must of necessity influence the medium upon which such activities depend. The effects may be shown upon the heart, the vessels, arteries, capillaries or veins; and upon the blood; that is, its quantity, viscosity, concentration or ability to transport respiratory gases."

Disturbance of any of these functions may be a factor in the development of gangrene or necrosis.

ETIOLOGICAL FACTORS IN PRODUCTION OF NECROSIS

Circulatory Changes.—It is found that thrombosis and embolism are the important factors in the production of necrosis and gangrene.

Physical and Chemical agents

(a) Pressure.
(b) Crushing injuries.
(c) Chemical agents, such as mineral acids, carbolic acid, bichloride of mercury.
(d) Ergot; possibly the long administration of ergot, causing, as it does, definite contraction of the arteries.
(e) Extremes of temperature, either heat or cold, are factors in the development of thrombosis.
(f) Electric and radio-active bodies, such as radium.

Bacteria and Their Products.—The most interesting part of my subject will be conditions which we believe have been caused by bacteria and their products. The associated trauma of fetal pressure, instrumentation and operative procedure should also be considered. Occasionally, the virulence of bacterial poison is so great that cells are killed outright, and a comparatively large area of degenerated tissue may be seen. Intense inflammation may cut off the blood sup-

ply, causing sloughing, and this may be caused also by direct action of bacteria.

Nerve Lesions.—Time will not allow discussion of the influence of nerve supply upon the organs under consideration. Abscess of the vulva and gangrene affecting the external genitals, and occasionally the cervix uteri, may be the result of direct injury affecting the deeper circulation, and the same condition may be due to violent instrumentation at delivery. In considering the changes which may take place in fibromyomas of the uterus, we should not overlook the possibility of necrosis or necrobiosis. In some cases necrosis may extend and produce similar changes in the associated body of the uterus. The same thought may be applied to strangulation of submucous myomas and polyps.

Improper irradiation may be a factor in producing necrosis and gangrene of the uterus. One case was seen in an eastern clinic last year, in which considerable destruction of the cervix, due to improper administration of radium, had taken place. Well defined intramural abscess of the uterus of puerperal origin, with the streptococcus as a cause, has been noted by almost all observers in gynecological work. But to general gangrene of the uterus arising from such localized abscess formation, there is practically no reference in the literature. With lymphangitic or thrombophlebitic sepsis, gangrene may occur and apparently no surgical procedure is able to benefit this condition. Bichloride of mercury gives protoplasmic poisoning and may kill tissue.

Goodell, years ago, advocated the use of zinc chloride in treatment of epitheliomatous masses in the uterus. More recently, papers have been presented by Drs. Babcock, Mason, Mayo, and others, calling attention to a revival of the use of this chemical; but I think this procedure has not met with general favor. Zinc chloride has always seemed to me a dangerous chemical. The effect when it is applied to the interior of the uterus cannot accurately be determined. It should be classed as a dangerous procedure. If the slough or destruction of tissue extends to the peritoneum, it may as well extend through the peritoneum, and be a grave menace to life. Before taking up, more in detail, the causes of gangrene, in which bacteria and infection play a large part, let me call your attention to the fact that a uterus which is not pregnant, or has not recently been pregnant, or has not been entered or traumatized by instrumentation is peculiarly resistant to invasion and infection by all microorganisms, except the gonococcus.

OFFENSIVE AMNIOTIC FLUID

The fact that amniotic fluid is occasionally very offensive led me to investigate the literature to determine whether abnormal amniotic fluid was indicative of a septic or necrotic uterine wall. Study of the available literature fails to reveal that the uterine musculature has been involved, although death of the fetus in several cases, and very long labor were the rule. Some of the mothers developed evidence of sepsis, which was followed by death. Unfortunately, no post mortem records were available in these cases. M. H. Jones, in looking over notes of 3,000 cases at the British Lying-In Hospital, London, found 5 cases of a peculiarly offensive condition of the amniotic liquor. In these cases the liquor was flocculent, brownish in color, muddy in consistency, and had an odor exactly resembling that of the contents of the rectum, very much like a typhoid stool.

S. Remy, of Nancy, France, reports 7 cases. In his series, the death of 4 children and 3 mothers was reported; and evidence of infection in other mothers was found.

J. Bailli, of France, reports two cases with death of two babies and the two mothers. In none of these cases was any mention made of the condition of the uterus.

Bacteriological examination of offensive liquor amnii developed the fact that bacteria were very numerous, mainly coliform bacilli. The extremely fecal nature of the odor suggested that infection from the neighboring bowel had, in some way, occurred, and it was highly probable that *B. coli* was the determining factor in producing the change. The bacteria may have been derived from the neighborhood of the anus and have extended up the vaginal canal, or may have passed directly through the wall of the uterus from the intestines, or may have extended down the Fallopian tube to the uterine cavity.

It is evident that infection of the amniotic fluid may occur without rupture of the membranes. The site of the placenta should be considered as a possible route of infection from the circulation.

From study of these cases it seems certain that infection of the amniotic fluid may exist without deep infection of the uterine musculature. In other cases it seems that there was maternal infection which could be attributed to an abnormal condition of the amniotic liquor.

I shall review 3 cases of gangrene of the uterus

under my personal observation and 9 cases selected from American, French, English and German literature. These were the most important ones in the literature to which we had access. I have made no attempt to go into the Russian literature, where the number of cases reported was very few.

CONCLUSION

American, English, French and German literature have references to gangrene. Probably the condition has not been recognized more frequently in this country because post mortem examinations are not resorted to here as frequently as in Europe. The presence of abnormal fetid amniotic liquor, which occasionally is noted, is at times associated with difficult or protracted labor, and in these cases, death of the fetus, and serious infection, with death of the mother may occur. In these cases gangrene of the uterus was not reported. However, post mortem examinations apparently were not done.

Death usually occurs when such a serious pathological state develops. The condition may exist with or without general peritonitis.

The causes are trauma in delivery, bacterial invasion, application of chemicals, radium, systemic circulatory changes as shown in one case, in which gangrene of the lower extremities was encountered, and necrotic changes in associated tumors of the uterus may result in necrosis of that organ.

No treatment is effective if the condition is well developed. Hysterectomy is indicated if evidence of necrosis is detected early.

CASE REPORTS

Case 1.—Bichloride necrosis of vagina, cervix and mucous membranes of mouth and gums.

Mrs. Annie Love, age twenty-five, entered Baylor Medical College Gynecological Department, Nov. 17, 1927, complaining of very sore throat, inability to swallow and muscular soreness all over the body to such an extent that she had difficulty in moving at all and sensations of chilliness and general discomfort.

She complained of frequency of urination, getting up ten or twelve times at night. At the time of her entrance to the hospital she had marked vaginal discharge and pain accompanying the act of urination. Three or four days before entering she was taken suddenly ill with chill and general aching. She could not rest, lying down or sitting up, had elevation of temperature, then a chill. She continued to vomit at intervals and was miserable. Her throat grew progressively worse for several days and burning in the vagina with a large amount of discharge continued. Her breath was very foul. She had two large ulcers and several small ulcers on the mucous membrane of the mouth. These were dirty gray in the center and white around the margins. Her gums were spongy and red and showed the same ulceration as the other mucous membranes. The tongue had white spots. The tonsils were enlarged and the right one was covered with a dirty gray membrane. The pharynx was hyperemic. Her blood pressure was 110/80. Abdominal examination was negative, heart and lungs were negative except that there was dypsnea. Vaginal examination revealed a dirty grayish membrane over the mucous membrane, and at the orifice of the vagina the cervix was covered with the same gray membrane. Her 'phthalein output was 46 per cent; 2,400 c. c. urine were voided in 24 hours. Urinalysis showed a trace of albumin and many pus cells with a few clumps. On November 17, 1927, blood count was: 3,700,000 red cells; hemoglobin, 75 per cent; white cells, 16,500; polymorphonuclears, 87 per cent; small lymphoctes, 10 per cent; large lymphocytes, 1; transitionals, 1; eosinophil, 1.

November 19, white cells, 13,900; polymorphonuclears, 80; large lymphocytes, 1; transitionals, 1. Wassermann and Kahn were positive. November 19 a special report was made on specimens of material from smears of the throat and vagina. They were examined by culture and direct smear. Direct smear from the vagina showed many leucocytes, a large number of long and short chain streptococci; a moderate number of staphylococci and a few diplococci, probably pneumococci. There were fusiform bacilli in the vaginal smear.

November 21, no diphtheria bacilli were found. Culture from the vagina showed a few colonies each of a streptococcus and a staphylococcus.

November 28, swabs were made from the cheeks and gums. No diphtheria bacilli were found in cultures from the cheek or gums. Both had identical cultures with many colonies each of a streptococcus, staphylococcus, *Micrococcus catarrhalis* and a gram-negative bacillus. On November 17 she was seen by a nose and throat consultant, who did not think this was a diphtheritic condition. She was also seen by two very competent dermatologists and syphilographers, who failed to make a diagnosis. It was suggested that she had Vincent's, diphtheria, or luetic involvement of the mucous membranes.

In going back over her history more carefully we found she had been accustomed to using bichloride douches, and a few days before coming to the hospital she took a concentrated douche, two tablets in a small amount of water. This was followed in a short time by chilly sensations, burning in the vagina, and a day or so later by the lesions in the mouth.

We had a stormy time with this patient. She lost a number of teeth. It was with difficulty that she secured enough nourishment. The condition in the mouth and gums responded very slowly to treatment. In the vagina the gray membrane did not disappear rapidly and the cervix resisted ordinary attention. After a number of weeks the mucous membrane had gotten back to a very much better condition.

Evidently the necrosis was due to prolonged use of bichloride, and the acute condition was caused by an excessive amount of mercury in the vaginal douche used a few days prior to coming into hospital. In this case there was none of the marked disturbance of kidney function usually found in mercurial poisoning.

Case 2.—Adis Brown, a colored woman, aged 23, (author's Gynecological service, Baylor Hospital), died five hours after entering the hospital from parametric septic infection following emptying of the uterus, by criminal abortion fifteen days before. She gave a his-

tory of passing blood and mucus from the vagina, but had had no frank hemorrhage before entering the hospital. She was restless, semi-conscious and obviously seriously ill. However, her temperature was only 98.8°, pulse, 110; respirations were 22; blood pressure was 110/75. The low temperature and other findings were probably due to her terminal condition. She had 15 per cent hemoglobin, 1,260,000 red cells, 45,000 white cells; color index, 0.6; 95 per cent polymorphonuclears; small lymphocytes, 5. Soon after she entered the hospital, hiccoughs and embarrassed respiration were noted, followed by death.

Autopsy.—The uterus about normal size and a trifle soft. The tubes and ovaries appeared normal. The uterus upon being opened seemed to contain placental shreds. The cervix was so incorporated in a shaggy gangrenous mass as to be hardly distinguishable. There was an abscess between the bladder and uterus with gangrene of the surrounding tissues. The smaller vessels of the uterus exuded pure pus. No thrombosis of the larger vessels was seen. There was no peritonitis.

The pathological diagnosis was:

(1) Acute suppurative endometritis and metritis.
(2) Parametric abscess with early organization.
(3) Acute non-suppurative interstitial nephritis.
(4) Cloudy swelling of the heart and liver.
(5) Acute splenic hyperplasia.
(6) Acute hyperplastic lymphadenitis.

Microscopical examination showed:

Uterus.—The stroma showed infiltration chiefly of lymphocytes, but with a few leucocytes, which increased in number as the endometrial surface was approached. The blood vessels were congested. The endometrial glands were absent, the surface was covered by a granular exudate in which structure is not visible. Between this and the muscle tissue was a zone containing abundant infiltration of leucocytes.

Case 3.—Mrs. J. O. Long, age 30, was seen by me on the service of Dr. Dexter Hardin, at Parkland Hospital. She was delivered in January, 1928, in this hospital of a still-born fetus, at term by high forceps. She remained in the hospital for one week after delivery. Nothing unusual was noted in her condition when she left the hospital. She had had four former pregnancies. After the delivery in January she had two periods, then missed. On June 4, 1928, she again entered the hospital with a history that three weeks previously she had had pain and cramping, passage of blood clots, followed by foul discharge of bloody material. Her temperature was 102°; respirations were 24; pulse, 100; white cells, 12,000; polymorphonuclears 70 per cent. The uterus was enlarged and nodular with necrotic tissues in the cervix. The odor of the tissue was very foul. The uterine cavity was quite open. The rim of the cervix had the appearance of carcinoma. Some of the necrotic tissue was removed eight days after admission. A few days later severe hemorrhage developed. While attempts were being made to control this hemorrhage, more tissue of a white membranous character was obtained. The cervix at that time was of much more necrotic appearance. The condition was grave and two blood transfusions were done.

The history would indicate that the patient had aborted before coming to the hospital and that membranes or a portion of the placenta had been retained. From this, septic infection developed with gangrene and thrombosis of vessels in the leg and infarcts in the lungs. The pathological report indicated that the first tissue presented was malignant. No definite opinion of the second specimen was given except that it was called necrotic tissue. The third blood transfusion was done when it was evident that the patient was absorbing toxins from necrotic areas. She was running a constant septic temperature to 103°. Twenty-two days after her admission to the hospital another attempt was made to remove some of this necrotic tissue. A large white membranous mass of tissue came out. The pathologist reported only necrotic material. The next day phlebitis developed in the right leg, followed by cyanosis of the right foot and evidence of phlebitis in the left leg. The plantar vessels were thrombosed. In three days, moist gangrene developed in the foot and extension of interference in the circulation of the left leg was noted. The condition of the patient was such that amputation was not considered advisable. Six days later embolus in the left lung, lower lobe, developed and there was evidence that the right femoral was occluded. Progressive sepsis ended in death thirty-three days after admission.

Autopsy by Dr. Hibbard.—Ecchymotic spots were found on the arms. The right leg was cyanotic and edematous. The omentum was densely adherent in the lower abdomen. The small intestines were glistening and congested. The peritoneum was shiny, the appendix small and free, the uterus and adnexa were gangrenous, walled-off from the intestines by omentum. The right ovary was hard and had the general appearance of malignancy. The left ovary was incorporated in the gangrenous mass. The uterus when freed from the broad ligament was easily lifted out of the pelvis, due to the extensive slough. It was entirely necrotic. The right lung, lower lobe, had a number of infarcts. The left lung had one sub-pleural infarct. The kidneys showed acute parenchymatous involvement.

PATHOLOGICAL DIAGNOSIS

(1) Gangrenous endometritis, metritis, and parametritis.
(2) Gangrenous salpingitis and oophoritis.
(3) Localized pelvic peritonitis.
(4) Thrombophlebitis, posterior tibial, and pelvic vein.
(5) Gangrene right foot and leg.
(6) Embolus of the pulmonary artery with pulmonary infarction.
(7) Cloudy swelling of the heart and liver.
(8) Acute hypoplastic splenitis.
(9) Chronic glomerular nephritis (slight).
(10) Hydronephrosis right, slight.
(11) Carcinoma simplex, ovary, bilateral.
(12) Secondary metastatic carcinoma of lymph node.

Case 4.—The following cases are from the literature designated:

Dr. Bichat's patient had an abortion of a four months' fetus four days previous to her entrance into the hospital. The abdomen was distended, hard, painful. There was every indication of an extremely grave condition. Abdominal section showed general peritonitis. The uterus was large and soft, with two large black plaques, with perforations into the abdominal cavity. Total abdominal hysterectomy was done, with death next day.

Case 5.—Dr. A. T. Weston reported on a post mortem specimen. Death of the patient occurred four days after a premature birth, following instrumental abortion. There was general septic peritonitis. There were gangrenous spots on the fundus and cervix.

Case 6.—(Dr. Lawson Tate). A patient, age 34, who refused treatment in the hospital for pelvic pain, had an offensive watery discharge from the uterus, which was soft and flabby. She died a few days later. The uterus was a jet-black, sloughing, stinking mass, with only about one square inch of normal uterine wall at the fundus. No opinion was advanced as to this serious infection.

Case 7.—(Dr. G. W. Phillips). Mrs. H., in labor, was vomiting every few minutes. There was abdominal soreness. A hand introduced into uterus found the ear of a child immediately above the pelvic brim, lying crossways, occiput to the ilium and face towards the left. The head and neck were curved and doubled up on the shoulder, and had not apparently been moved at all by the labor pains. Manipulation failed to effect delivery. Death followed that day, without delivery. Post mortem showed the placenta and major part of uterus in a state of mortification. The peritoneum was inflamed as was also one-half of the surface of the child's body. The stench was terrible.

Case 8.—(Dr. Aumont, Paris). The patient, age 44, had very severe uterine bleeding. Her general condition was alarming, her pulse hardly perceptible. There was evidence of fibromyomas. The uterus was soft, and painful. A large, fibrous, degenerated polyp was lying in the vagina. A little later during a temporary improvement of the patient, a large polyp was removed, which had a repulsive odor. The uterine wall was blackish gray in color. Later the condition of the patient improved somewhat, but it was found that she had another fibromyoma high up in the uterus, so a vaginal hysterectomy was done. The uterus was found to be superficially gangrenous; also several pockets were found. Recovery was accompanied by nephritis and was rather stormy.

Case 9.—(Dr. Keller, Paris). The patient, age 33, had a spontaneous abortion at the fourth month, followed immediately by a chill, vomiting, painful abdomen, and evidence of extensive peritonitis. The uterus was large and sensitive, with a very fetid discharge. Immediate laparotomy was done. The appearance of the uterus was most unusual. The fundus had an area of blackish gangrene from which bubbles of fetid gas were escaping. The left adnexa were black and large, the vessels in the broad ligament thrombosed. The patient died several hours later. The uterus was a black pulpy mass up to the peritoneum. *Bacillus coli* and *Bacillus perfringens* were present.

Case 10.—(Dr. Stanka, Germany). In a patient age 19, spontaneous amputation of the cervix occurred after gangrene of the isthmus in the course of pregnancy. Abdominal pain was noted, and frequent vomiting. She had not menstruated for four months. She was unconscious when she entered the hospital. The abdomen was distended, the tongue dry, limbs cold, vagina shortened, fixed and swollen. Death occurred two days later. At autopsy, the peritoneum did not show the slightest inflammation. Infected liquid was found in the right parametria, and foul fluid below the uterovesical peritoneum. There was a fetal skeleton in this area. The uterine cervix was completely severed and gangrenous. In the uterine cavity nothing abnormal was found. The entire surface of the injured area was covered with necrotic tissue.

Case 11.—(Dr. Oastler). Spontaneous gangrene of the cervix was reported. The patient gave a history of having had previously a severe acid burn on the wrist, which caused a large slough with extensive necrosis of the edges. The wound was treated by skin grafts, which healed very slowly. A few broke down. A little later she had a similar wound over the crest of the ilium, following a slight injury by a corset stay. As there was a vaginal discharge, examination was made and revealed a similar condition with the cervix involved in vesicles and areas of necrosis. The necrosis was probably caused by the same pathological condition which caused necrosis of the skin. Infection may have been borne by the circulation.

Case 12.—(Dr. Weinzier, Berlin). The patient, age 21, was a primipara, at short term, with a narrow pelvis, prolapsed cord, and fetus dead by asphyxiation. She had had a forceps delivery. She developed peritonitis ten days later. Laparotomy showed gangrene of the uterus, which lay entirely detached below the serosa. The adnexa were deep red in color and swollen. The uterus was a dark green, sponge-like mass of gangrene, with several openings in its wall. It was removed without loss of blood.

In looking over the available literature of gangrene of the uterus one notes that there are relatively few cases reported. In all probability this is true because post mortem examinations have not been generally made. But reference has been made rather indefinitely to a number of cases, probably 60 or more. A greater number of these cases seem to have been in Russia in the period 1880-1900. Keller thinks that anaerobic microorganisms, which are found in normal conditions in the vault of the vagina, and in the cervix and intestines, are responsible for this infection, which produces necrosis. He thinks, also, that these microorganisms may cause a comparatively benign infection of the amniotic fluid and uterine cavity in long deliveries. They may provoke a gas, giving what is called uterine tympanites. This is thought to be much more common than uterine gangrene. He is also of the opinion that mixed infection may allow common bacteria to prepare the ground by disintegration of the placenta, membrane and blood clots, and that on this tissue anaerobes more easily develop.

The very rare condition, cervical pregnancy, may produce necrosis of the lower segment of the uterus. Rupture of the uterus, either spontaneous or caused by instrumentation, may produce necrotic areas, but this condition will not be considered in detail in this paper.

It is a matter of record that complete cervical ring-shaped separation has occurred after septic gangrene has developed. Gangrene of the uterus most frequently occurs as a complication following criminal intervention, a few following delivery at full term, the labor having been long drawn out, and delivery associated with trauma.

Weinzier's records show that necrosis may be present a very few days after abortion or deliv-

ery, but it usually occurs about the fourteenth day and often as late as the thirtieth or fortieth day. Recovery takes place with severe scar formation, and the uterus often becomes completely atrophic and atretic with permanent amenorrhea and sterility. The death rate is considered to be about 30 per cent or more, and death occurs under symptoms of progressive general sepsis, or unexpectediy and suddenly due to peritonitis.

All who have described this condition or written upon this subject, agree that early diagnosis is difficult and usually impossible. The treatment should be very early and probably hysterectomy offers the greatest opportunity of recovery for the patient.

REVIEW OF 12 CASES OF GANGRENE OF THE UTERUS

	Total No. Cases
(1) Peritonitis	4
(2) Chemical agents used	1
(3) History of preceding abortion	6
(4) Delivery of fetus	3
(5) Laparotomy performed	5
(6) Other operations performed	2
(7) Fibroids or polyps present	2
(8) Adnexal necrosis	4
(9) Separation of cervix from body	1
(10) Gangrene of lower limbs	1
(11) Parotid gland infected before delivery	1
(12) Cervix alone necrotic	3
(13) Uterine body necrotic	9
End Results	
(1) Death of fetus	2
(2) Death of mother	8
(3) Recovery of fetus	2
(4) Recovery of mother	4

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DISCUSSION (*Abstract*)

Dr. W. R. Cooke, Galveston, Texas.—Gangrene in fungating cancers, particularly of the cervix, and occasionally in endometrial cancers, is quite common. In subserous fibromyomata we see it occasionally. In the uterus proper it does not often occur. The blood supply to the uterus comes from so many sources that it is difficult to shut off either the arterial supply or the venous drainage. Theoretically, the etiology of gangrene of the uterus as a whole should be limited to the factors which would obstruct all the blood supply to the uterus, as in a universal thrombosis.

In looking over the material in the records of the John Sealy Hospital covering thirty-nine years, we were able to find only two cases of true gangrene of the uterus. One case followed a septic abortion. The history is incomplete, but the specimen is in the museum. In this case there was a definite thrombophlebitis extending out of the uterus, along the veins into the broad ligament, and involving all of the veins on the left side. The other case followed the taking of oil of tansy. In that instance there was also complete gangrene of the mucosa of the small intestine and, to a less degree, of the entire gastrointestinal tract.

Necrosis of uterine fibroids, particularly the intramural type, is a common finding, but true gangrene in the intramural fibromata is quite rare. In the obstetric field, we used to get a good many women from Mexico who had experienced absolutely neglected dystocia. These women used to come in in very bad condition with huge vesicovaginal fistulae due to pressure necrosis and gangrene; but it was notable even in such cases that the uterus was less affected than any other part of the genital tract. In the case which followed the

taking of oil of tansy there was more gangrene in the vagina than in the uterus proper. We had one case of bichloride poisoning in which the uterus showed considerable necrosis (hardly enough to call gangrene) in the endometrium and the inner parts of the myometrium.

Perforation of a pregnant uterus will sometimes bring about gangrene, but there again it is more marked in the surrounding structures than in the uterus proper.

We have had two cases in which gangrene in a mild degree was present in senile evolution changes. In these the factor of infection was present, and the infection probably played the greater part in the gangrenous process.

Dr. John W. Turner, Atlanta, Ga.—I have seen one case of gangrene of the uterus. It occurred after abortion in an Atlanta woman, who had used a catheter with a wire. The patient came to me after she had gone home and developed a pelvic peritonitis. It was necessary to operate and we found the fundus of the uterus gangrenous. The wire had perforated the uterine wall. She finally recovered, but gave us a good deal of worry.

Dr. G. E. Johnson, Ardmore, Okla.—I have seen only one case of true gangrene of the uterus, and this followed an abortion and bad laceration, with, in addition, a severe tubal infection. Since there was no postmortem the degree of gangrene was not determined.

The next case, which was at first diagnosed as gangrene, proved to be gangrene only of the placenta. This patient had passed her date of confinement two months, having fever, rigors, and a very violent discharge from the uterus. She was delivered of a necrotic fetus and a rotten placenta. All symptoms cleared up within a few days, and the patient, I am glad to say, made a rapid recovery.

Dr. Lawrence R. Wharton, Baltimore, Md.—I saw an interesting case in the last few years of localized necrosis which followed the use of radium. The patient was a young girl, about sixteen years old, who had been irradiated for hyperplasia, an entirely benign condition, and she did not menstruate after that for two years. She then began to menstruate slightly and nine years later came to Baltimore to have a curettage because of profuse bleeding. When I passed the curet it went right through the uterus without any resistance at all. I curetted around this place and put in a small drain. She had a little pelvic peritonitis for a few days but no other difficulty. Her menstrual periods became normal and remained so for about six months, when she again had bleeding and returned for further treatment. We then removed the uterus and found a deep area of necrosis in it. On the posterior wall there was an area which had been completely destroyed by the radium. There was nothing but peritoneum and omentum over this area on the posterior wall, which of course, was the first spot one would strike in a curettage.

I mention this case as something to be remembered when using radium in treatment.

Dr. Dunlap (closing).—In the old literature a good many cases of uterine gangrene are reported and as we have shown here today, each man now remembers a few instances. I am sure there have been a number of cases in America which would fall under the head of gangrene or necrosis of the uterus, but I have been unable to find them in the literature.

PELLAGRA*

By J. T. MARTIN, M.D.,
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Pellagra involves three distinct systems, each in a striking manner. The skin is affected with a defacing dermatitis; the digestive system is disturbed with a debilitating diarrhea; and the nervous system supplies symptoms varying from a mild neurasthenia to a distressing dementia. The medical profession of our country has contributed richly to the history of the fight against the ravages of this scourge. The increase in the prevalence of this disease in Oklahoma is the occasion of this review. And while other states of this Association have longer and more extensive experience, we of Oklahoma have a real problem.

In 1735 was written the first paper on pellagra. A Spaniard by the name of Gasper Casal at this date wrote of the disease in Oveido, but did not publish his observations until nearly thirty years later. It was observed in Italy even earlier and an Italian by the name of Prapolli is credited with the naming of the disease. The name "pellagra" probably means rough skin. In 1784, at Legnano, was established the first recorded pellagratorium. France, which today has little pellagra, furnished the next writer, one Hameau, whose observations began in 1818 and were reported in 1829. Prumer, in Egypt, described the disease in 1847, but there is no further discussion from this pellagrous country until 1893.

Pellagra is first mentioned in our country in the Northern states. In 1864, Dr. John Gray, of Utica, New York, and Dr. Tyler, of Sommerville, Massachusetts, each reported a case. An Italian sailor furnished Dr. Shewell, of Brooklyn, a case in 1883. Charity Hospital records show a case in the service of Dr. Bemis in 1889. The disease doubtless existed in the South before, but was not recognized as an entity. Records in the hospitals of the South as early as 1834 seem to establish this as a fact.

An intensive study of the disease in this country followed the report of Searcy in 1907. This report was of an epidemic in an asylum in Alabama. The disease was reported from all over the South in a succession of startling findings until the entire profession of the South and the Public Health Service was aroused. The challenge was accepted. A galaxy of names, men who did a stupendous labor, was written in the pages of American medical history.

Foremost among these was Joseph Goldberger. Together with his co-workers of the United States Public Health Service and the physicians of the South, he, in the next twenty years, carried on one of the most remarkable studies of epidemic disease that medical history records. His efforts clarified many of the factors causing the disease and standardized the treatment. But the conquest is not complete. Suggestions for the pre-

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vention were given, but prevention has not been accomplished. Greater knowledge and the spread of this knowledge and perhaps a better enlistment of the economic forces of the community are needed. Much remains to be done.

Geographical Distribution.—Pellagra is found in 80 per cent of the United States and in all her extra continental possessions. The lower Mississippi Valley, the states of the Southeast and the states of the Southwest are the areas of the greatest prevalence. In Europe, the Mediterranean countries and the Balkans are the areas of greatest prevalence. Asia has many pellagrous regions. The best known are those of Asia Minor and India; South Africa, the Red Sea and the Mediterranean seacostal countries of the Black Continent are subject to endemic pellagra. South American countries report some cases. Goldberger, shortly before his death, estimated that in four years the increase in the Mississippi Valley would be 100 per cent. Dr. Charles Laughinghouse, in an account of a conference of members of the United States Public Health Service this year, reports an alarming increase of this disease. He reports 301 new cases in July for North Carolina alone; the same State in June had 122 deaths and in May 115 deaths. The total deaths for the first six months for the State was 468 out of 1,442 cases reported. The total afflicted in the State is estimated at twenty thousand. The Oklahoma State Health Department likewise reports an increase, and so do all the states of the endemic area. It is estimated that in the Mississippi Valley there are from forty to sixty thousand pellagrins. With so wide a distribution and with nearly all areas reporting an increase in the incidence and the death rate of the disease, pellagra becomes of major importance both as a medical and as an economic problem.

Hard times increase the prevalence of pellagra. This is easily understood, as poverty is a vital factor in the causation. Less than 4 per cent of cases occur among the well to do. What will the present agricultural and industrial depression do to the incidence of the disease? The test of our theories may be at hand.

The maize theory of the cause of pellagra is the oldest. Marzari, in 1810, first mentioned the possibility of the relationship of maize to pellagra. This idea has been studied and followed by many and is still commented on in the literature. Early this year, Dr. H. B. Day says that "Pellagra bears the same relationship to maize that beri-beri does to rice." Maize is considered by the later students as a predisposing rather than an exciting cause.

Living organisms have often been assigned an etiologic role in this disease. About two years ago Terrell and Venable read a paper before the Oklahoma State Medical Association on complement fixation test by prepared antigen for diagnosis, and supported the idea of an infectious cause. Dr. Francis, of the Public Health Service, after making 94 animal inoculations of every kind of material from a pellagrin, obtained negative results. He concluded that his work gave no support to the theory of an infectious cause. There is no convincing evidence today of the infectious theory.

The meat protein theory is heartily supported by the English writers. W. H. Wilson, of Cairo, is a strong supporter of this idea. In the *British Medical Journal* of January 18 this year, Wilson cites the Dunbar Prison outbreak studies in support of the idea. Professor McGraw, before the Section on Tropical Diseases of the Royal Society of Medicine, affirms that the "vitamin theory has not been established to such an extent that all other theories are put out of court."

The vitamin theory was urged and developed by Goldberger. It was first hinted at by Marzari and this theory leads all others in the number of its followers. With this theory there are two ideas prevalent, the first that the absence of a vitamin, P. P. or B_2 or G causes the disease. In Goldberger's experiments at the Mississippi State Penitentiary near Jackson, Mississippi, 6 out of 11 prisoners developed pellagra when fed on a diet that was low in vitamin P. P. and low in meat protein. The same worker conducted an experiment at Jackson with two orphanages, neither of whose inmates developed a case of pellagra when on a diet that was rich in vitamin P. P. and in meat proteins (Goldberger and Waring), although at both institutions pellagra had previously been endemic. At the Georgia State Sanitarium a samilar result was shown in which the usual incidence of pellagra prevailed among the controls while the disease was absent from those on the anti-pellagra diet. In his last effort Goldberger satisfied himself that meat protein was not essential if the vitamin was present: that is, that pellagra was caused by the absence of vitamin P. P. and this vitamin, while found freely in meat, was present in still richer quantities in yeast.

Dr. H. B. Day, in the *British Medical Journal* of January 7, this year, suggests that the absence of vitamin P. P. causes atrophy of the intestinal mucosa and that the ensuing mal-digestion and mal-assimilation complete the vicious circle. Dr.

R. B. Ellis, writing on the subject of pellagra secondary to gastro-intestinal diseases, expresses a similar idea.

The Oklahoma State Board of Health reports an increase of 134 per cent in pellagra deaths between 1925 and 1929; the cases reported increased in the same years 90.75 per cent. They also assured me that the report for this year when compiled will show as great or a greater per cent of annual increase. Our state sanitoria show a similar increase. At the Eastern Oklahoma hospital the increase in four years is 150 per cent. At the Central State Hospital the increase in admissions for the fiscal year ending July 1, 1930, was 33 per cent more than last year. In Oklahoma City at one of our private hospitals (St. Anthony's), which has few ward patients, one-third more cases were admitted during the first seven months than during any previous year. This percentage projected into full years would represent 233 per cent of the previous high year, or an increase of 133 per cent. The admissions of pellagrins at the University Hospital for the first six months of the year were two-thirds of the total admissions for all of last year and more than double that of any year between 1921 and 1927. The deaths at this institution for the first six months were exceeded for the entire year only twice in the last decade, namely: 1921 and 1928. The percentage of mortality of all cases was 18.3. The histories show forty different complications listed, of which eight were referable to the gastro-intestinal tract. Of all admitted cases, 15 per cent had gastro-intestinal complications. In 1929, the gastro-intestinal complications represented 16 per cent. In 1930, due to closer study, 40 per cent were found to have such a complication. These complications were exclusive of diarrhea and absence of gastric acidity. Absence of hydrochloric acid was found in 60 cases, or 6 per cent of the cases checked. *Endameba histolytica* was found in 29 per cent of the cases checked. Unfortunately the number of cases in which the search for intestinal parasites was made is small. The work has been done mainly in the last two years. Drs. Ely and Keltz devoted much time to the study of amebiasis in pellagra and they have demonstrated repeatedly the parasite or cysts in smears taken through a proctoscope and examined immediately, even when stool examinations were reported negative. We expect to see the percentage of amebiasis reports among our pellagrins increase by the use of this method of search for the organism. It may be that, as Dr. Ellis suggests, the problem of pellagra control is in good part control of the complications. At least, the complications, especially those suggestive of intestinal parasites, deserve a more careful study, because the secondary infection might be the agent that precipitated the deficiency and allowed the attack.

SUMMARY

(1) The distribution and number of cases of pellagra make it a disease of economic and medical importance to the South.

(2) The increase in pellagra despite our increased knowledge is a direct challenge to the profession of the South.

(3) The present economic distress doubtless will increase the menace to our communities.

(4) To eliminate this scourge is probably the greatest work of community service before the profession today.

(5) This Association should lead in the effort.

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DISCUSSION (*Abstract*)

Dr. V. S. Caviness, Raleigh, N. C.—The question of pellagra interests any physician practicing in the southern part of the United States and in almost any subtropical country in the world. You noticed from the essayist's paper that the distribution of the disease includes practically all of the subtropical countries that are pretty well civilized.

A number of us who have been studying this disease look upon it as due to some infection by a tropical or subtropical organism. That has not been proved. With reference to diet, Goldberger did all his work and spent his life trying to prove that it was a vitamin deficiency disease. However, that has not been proved.

The essayist spoke of an increase in pellagra in North Carolina during this year. It may be of interest to you to know that one of the greatest increases in North Carolina has occurred in the negro insane asylum where the diet is as good as it can be and where they see that the patients get plenty of vegetables, plenty of vitamins and everything else to prevent pellagra.

Another thing of interest is that you find one, two or three cases in one family and the remainder of the family not having the disease, although all the family were on the same diet. Another item which I consider

somewhat against the dietary argument is that we see it in children. Just this year I had a case in a child, three years old. It occurs in children living on practically a complete milk diet, in good homes where the diet is very satisfactory.

There is quite a resemblance between this disease and pernicious anemia. Both diseases have either a total or practically complete absence of free hydrochloric acid in the gastric secretion. The absence of hydrochloric acid allows any infection in the mouth to pass from the stomach into the intestinal tract and there to exert characteristic influence. With one exception in a very small child all cases of pellagra that I have examined have shown badly infected mouths. They have all showed spirilla in the gums and also amebae, which I have not positively identified. However, finding the same organism in all cases I consider very suggestive. I hope to make a further report at a later date.

Dr. J. J. Gable, Norman, Okla.—All of us who live in the South are interested in pellagra, and I am of the opinion that next spring we shall be even more interested. I happen to be a physician in a state hospital where during the past few years we have treated some 500 cases of pellagra. I have noticed that in this hospital we seldom get a case of pellagra until it is in a far advanced stage. I wonder why we cannot make our diagnoses earlier. I wonder how many lives could be saved if the country physician knew just how to treat this disease. I do not intend to try to embarrass anyone, but it is almost alarming to see a pellagra patient come into the state hospital with all the bottles of medicine that the family physician has given him to take for pellagra. He has not been educated as to how to live, what to eat and how to sleep, to prevent these classical symptoms. We treat a pellagrin in our state hospital just like a tuberculosis patient, by absolute rest in bed, until all signs of intestinal and skin activities have ceased. We feed him a light diet with rare beef steaks and yeast, if there is not too much intestinal upset, but the main thing we try to institute is *rest*.

Until the physician knows how to diagnose pellagra in its incipiency, and until he ceases to treat the disease with medicine alone, until the county health units take greater steps along an educational line to prevent pellagra, until they teach the people what to eat and how to rest once the symptoms are developed, it is my opinion that great strides toward eradication of this dread disease will not be made.

Dr. W. W. McKibben, Miami, Fla.—I would like to speak from a pediatrician's point of view on pellagra, a disease which we rarely saw in New England, and which is extremely infrequent among children in southeast Florida.

This winter, however, I saw two cases of so-called acrodynia or Swift's disease, which, since the two autopsies performed by Warthin, has been referred to by Dr. Royster of the University of Virginia, and others, as the infantile type of pellagra. From the postmortem findings, Warthin feels that the disease is not a polyneuritis, erythredema, nor is there evidence of a specific infectious etiology, and there is no relationship between tonsillar disease and the syndrome. The changes in the skin and central nervous system, Warthin thinks, suggest the pathology of the early erythematous stage of pellagra. The entire anatomic picture suggests either a food deficiency or a toxic state acting on persons of the hypoplastic constitution, and affecting the reticulo-endothelial system, the meninges and the skin, the vegetative nervous system and possibly leading to a light sensitization. It is because of these findings of Warthin, that I, too, feel that acrodynia or pink disease is a disturbance of nutrition in which there may be a deficiency of certain food elements or vitamins, but believe that frequently these follow an upper respiratory infection of the kind that we speak of as intestinal influenza. In both of my cases there had been an excessive amount of sugar in the diet, finally resulting in a hyperacid condition of mucous colitis, scalding and burning of the buttocks, almost complete insomnia, erythematous rash, and pulling out of the hair. It is probable that my cases had insufficient protein in their diet at eighteen months, having had no beef juice, no scraped meat, no eggs, no raw nor scalded milk, only pasteurized grade A subjected to low heat over a long time; no cod liver oil, but viosterol only. Both cases responded in from ten days to three weeks to a proper balanced ration including all the vitamins and lactic acid milk, sufficient protein and a low sugar intake. In both instances all the symptoms had disappeared, but in neither instance could I obtain proper gain in weight until I took out the tonsils and adenoids.

In conclusion I believe that all three theories are correct, the etiological factor being an anaphylaxis similar to that seen in eczema, and a deficiency disease which in most instances follows a toxic state, acting on children of hypoplastic constitution.

KERATITIS: NUTRITIONAL, NEUROTROPHIC, DYSTROPHIC*

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I.—NUTRITIONAL KERATITIS

In making this somewhat didactic classification of certain corneal lesions and diseases, I may be accused of taking a poetic license. Yet, when one studies carefully the characteristics and course of many diseases of the cornea, he finds that they fall most logically into one of these classes.

In the first group I have placed phlyctenular disease, trachoma, xerosis xerophthalmia and keratomalacia, because in each it is the keratitic complication which causes such disastrous consequences, and because in each there is an addition to other etiological factors, a background of profound nutritional deficiency.

In our State and City, phlyctenular keratitis causes more suffering and impairment of vision than any other one ocular disease. The negroes are especially susceptible and usually have it in its more serious forms. Among negroes it occurs and recurs in all ages from the infant to the old men and women of sixty. Cridland[1]

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stated that 9 per cent of eye conditions in certain hospital centers in England were phlyctenular. In this same country, also, Hird[2] stated that where serious impairment of vision was due to corneal lesions 50 per cent resulted from phlyctenular disease. In our section, I am sure the incidence of visual impairment due to this disease runs equally high. The lesions of phlyctenulosis from the mild conjunctival efflorescence to the terrible phlyctenular pannus are so well described by various writers and are so familiar to all observers that I omit the classification.

In a review of the literature, one is astonished at the many theories, some visionary, some plausible, advanced as to the cause of phlyctenular outbreaks. Many theories have come and gone, influenced by the changing thought in the evolution and advancement of medicine.

(1) Earlier writers, notably Gifford[3] and Weeks,[4] were inclined to the bacterial origin. Gallenzo[5] claimed to have produced the disease by a certain micrococcus.

(2) The tuberculous theory has always had many adherents. It gained increased impetus after the cutaneous and other tuberculin reactions were discovered. The fact that the reactions were positive in such a large percentage of cases caused Fuchs, de Schweinitz, Wilder,[6] Derby[7] and many others to subscribe to this belief. Cridland inclines to the tuberculous idea. Quoting Priestly, this author thinks malnutrition and not environment increases the incidence. All the proponents of this theory agree with him in believing that phlyctenular disease is not tuberculosis *per se*, but is due to a tuberculous process somewhere in the body. Cridland, however, in spite of his tuberculous inclinations, lays great stress on diet, reducing carbohydrates and increasing vegetables, fresh fruits, and meats.

(3) An unbalanced diet with excessive carbohydrate intake has been considered by many careful observers as a cause. After a study of 500 cases, Goldenberg[8] came to this conclusion. In these cases he was convinced that tuberculosis played no part in the etiology. A more recent article by him[9] gives a most logical argument against the tuberculous origin of the disease. In the last article he emphasizes the value of the high vitamin diet.

(4) Many believe that autointoxication should be recognized as the chief offender. Bruns,[10] in a convincing article, says: "Phlyctenular ophthalmia should be regarded as a neuropathic phenomenon brought about by autointoxication." Theobald[11] held similar views.

(5) Later, focal infection as a cause climbed to the ascendency, especially focal infection due to teeth and diseases of the nasopharynx. Turner[12] states that phlyctenulosis is a disease of childhood and is due to a direct extension to the eye of a low-grade ethmoiditis. Facts, of course, controvert his contention.

(6) Lastly, anaphylaxis must not be overlooked, even though the experiments to date are not conclusive. Wolf[13] considered phlyctenulosis a symptom complex having its origin in the hypersensitiveness to toxins of the tubercle bacilli. His treatment was based on decreasing the sensitiveness of the eye by the use of tuberculin. Funaishi,[14] by injecting various proteins, including tuberculin, rendered a series of rabbits hypersensitive to proteins. Then, by instilling the reagent into the conjunctival sac of the sensitized animal, he was able to produce a condition resembling phlyctenular keratitis. He considers then that the disease results from protein hypersensitiveness. He was among the first to use boiled milk injections in its treatment.

My study of patients, my observations of the course of the disease and my experience with its treatment have forced the conclusion that it is essentially and directly the result of a basic disturbance of nutrition. Not all patients under similar conditions will have phlyctenular keratitis. The patient so affected, therefore, must have a constitutional predisposition. In such patients a perverted nutritional balance will excite manifestations. Tuberculosis, focal infection, deficiency diet, autointoxication, unhealthy surroundings, all play important roles as contributing causes. Regardless of tuberculosis or focal infection, time and again I have seen patients at Charity Hospital, New Orleans, who were suffering from severe phlyctenular keratitis clear up rapidly under the influence of high vitamin diet, sunshine, and fresh air. As long as we can keep them under these conditions they remain free from phlyctenular outbreaks. Remove the focal infection, treat the tuberculosis, clear up all systemic diseases; yet, when the patient goes back to his sordid surroundings and improper and insufficient food, he again becomes a victim and a repeater at the clinic.

Superficial vascular keratitis, or pannus, is primarily a part of the trachomatous process. Its early relief is the most important factor in reducing to a minimum the impairment of vision resulting from the disease. No disease has been written about more than trachoma. It is astounding how little thought has been given to

the nutritional aspect of this world-wide problem. Because of this fact I make bold to discuss it here. In 1926 Dr. Stucky,[15] before the Southern Medical Association, said: "I believe genuine trachoma will eventually be proven a deficiency disease, a term which is sanctioned by custom and embraces disorders due to faulty or unbalanced diet." The trachoma we see in New Orleans is not especially contagious. It is rather the rule to find the disease only in one or two members of a large family. This is in accord with Dr. Stucky's findings in the mountains of Kentucky.

In recent years our clinic patients are hospitalized, put on a high vitamin diet, as suggested by Dr. Stucky, and given the benefit of plenty of fresh air and sunshine. The standard local treatment is used. The result is: the pannus disappears early; in three to six months we can discharge these patients as cured, and they depart with useful vision, provided they have come under observation reasonably early in the course of the disease. In former years we treated them as out-patients. The victims were permitted to return to their homes to poor food and unfavorable environment. The course of the disease was long drawn out; the exacerbations many; the end results were most unsatisfactory. In many instances patients gave up in disgust, much to our personal satisfaction and comfort. To regard trachoma as a deficiency disease, until its specific virus is discovered and to treat it as such, constitutes a great step forward in the conservation of vision and relief of human suffering.

Xerosis, xerophthalmia and keratomalacia really represent different stages of the same disease. Unquestionably, this is the purest type of deficiency or nutritional disease. It is primarily due to lack of foods containing the so-called fat soluble vitamin A. This disease occurs: (1) during great famines; (2) in children profoundly ill from intestinal diseases. The milk, vegetables and cod liver oil may be given in abundance, yet absorption fails to take place from the intestinal tract; (3) in adults suffering notably from malignancy of the liver or pancreas, or both, the proper emulsification and saponification of fats cannot take place and the fat soluble vitamin A cannot be absorbed. The intake of food can therefore serve no purpose.

In such cases Blegvad[16] reports that he has obtained remarkable improvement of xerophthalmia complicated with severe night-blindness by giving parenteral injections of a concentrated fat soluble A, made from cod liver oil. He injected 2 c. c. of the concentrate every second day, with the result that the xerosis vanished, the corneal symptoms were cured, and the night-blindness was much improved. The technic for making the concentrated vitamin A is given fully in his article. This statement should prove of great value to those marasmic children threatened with blindness from keratomalacia due to malnutrition or severe intestinal disease. Naroz[17] discusses xerophthalmia as studied by the corneal microscope. Xerosis due to avitaminosis always shows cholesterin crystals in both the conjunctiva and the cornea. This phenomenon results from the fact that the reserve of fatty tissue is exhausted. Degeneration and destruction of cells and a solution of the fixed fats takes place. The cholesterin deposits are a logical consequence. Pillat[18] discusses the presence of xerophthalmia in adults in China. His article shows clearly its relationship to improper food. In America one rarely sees primary xerophthalmia or its malignant form, keratomalacia, except in individuals, mostly infants, who are profoundly ill.

A form of simple xerosis often seen by ophthalmologists is characterized by a sensation of dryness, a feeling as if the eyes had sand in them, or perhaps a foreign body under the lid. Examination reveals only slight irritation. Strands of mucus cling tenaciously to the cornea; breaks are noted in the epithelium. These change from day to day. The condition may be easily overlooked and treated locally. The following case illustrates my point:

Mrs. H., age 39, complained that her eyes felt dry and scratchy. Examination showed pallor of the conjunctiva; strands of secretion adhering to the corneal surface; a slightly reduced sensibility of the cornea; multiple breaks in the corneal epithelium, changing from day to day over a period of weeks. Local treatment did not influence the disease in the slightest. A careful history showed that the diet was deficient in fats and fresh vegetables. A corrected diet resulted in a prompt cure. All symptoms manifest in this disease are due to diet poor in food containing vitamins A and B.

II.—NEUROTROPHIC KERATITIS

In the neurotrophic group, I shall refer to the following diseases: (1) herpes simplex; (2) herpes zoster corneae; (3) keratitis punctata sub-epithelialis; (4) recurrent erosion of the cornea; (5) recurring primary bullous or vesicular keratitis; (6) neuroparalytic keratitis.

(1) Herpes corneae may assume various forms. One is the typical dendritic keratitis, or the simple ulcer with irregular outline. The herpetic lesions may be multiple and confluent so that a large area of cornea is denuded of

epithelium. Herpes corneae may occur as a local corneal reaction to the herpes virus without systemic affection. It may occur as a similar reaction to herpes virus when the body resistance has been lowered by some acute infection, notably influenza. I saw the greatest number follow in the wake of an epidemic of dengue fever.

(2) A less frequent neurotrophic reaction to the herpes virus is the keratitis punctata subepithelialis. In that type there appear few or many grey-white subepithelial punctate opacities in the cornea, singly or arranged in groups. Usually, they are seen in the center of the cornea, but infrequently they are peripheral. This type of keratitis commonly begins with an attack of conjunctivitis which soon subsides, to be followed in some days or weeks by the characteristic opacities. These may come and pass quickly to recur no more. Wright[19] gives a detailed account of an epidemic of this disease in Madras, India. As in herpes, recurrences may take place over a period of years. For example:

A young woman was seen in 1919 because of inflammation of her right eye. She gave a history of two previous attacks at intervals of four years. The attacks began with the usual conjunctivitis, followed later by the corneal lesions which remained for some months, disappearing, as a rule, when her general health improved. Again, in 1921, the fourth attack began in March. The opacities did not fade until the following December. Another mild attack lasted from April 12, 1924, to July 24, 1924. The disease and recurrences were confined to the right eye.

Case 2.—This demonstrates the recurrences of herpetic keratitis confined to the right eye during a period of thirty-one years, 1899 to 1930. Some attacks were mild, others severe. The patient had an attack of dendritic keratitis in the right eye following malaria in 1899. A second attack in the same eye, 1904, followed the grippe; it lasted one month. The third attack, in 1908, lasted 25 days. The fourth attack took the form of a herpetic ulcer in 1915, which lasted 58 days. In 1924 came the fifth attack, a dendritic type, without preceding illness. There had been no preceding illness, but a great sorrow—the loss of her husband. Two weeks ago the right eye became uncomfortable. Examination showed diminished sensibility and a few punctate breaks in the epithelium near the lower limbus. Diffused corneal opacities due to the many previous attacks reduced the vision to 20/40. (This case is reported from case reports of Dr. H. Dickson Bruns.)

In my experience, simple herpetic keratitis is unilateral and only rarely bilateral. These case reports tend to prove that whatever the character of the herpes virus may be, some individuals are virus carriers and "on the least lowering of tissue resistance, local or general," there develops a herpetic manifestation.

(3) A kindred neurotrophic disturbance is primary bullous or vesicular keratitis. Its pathogenesis is obscure, but it is unquestionably a lesion of the terminal branches of the fifth nerve and may be due to a virus with a dermatrophic tendency. This rare disease is said to result from slight abrasion of the epithelium. In many instances it seems to occur without an antecedent injury in perfectly healthy individuals.

In a woman, age 45, an attack began suddenly in 1925 in the right eye. Over a period of eighteen months, more than 25 bullae or vesicles appeared in various parts of the cornea, more frequently, however, in the center. The condition became quiet after two years with vision reduced to 20/200, due to permanent corneal opacity. In the early part of 1928 the left eye became similarly affected; there were recurring bullae and the resultant erosions. This case was seen by me in consultation in 1928, during an attack in the left eye. Her oculist was never able to find a cause. The disease, when I saw the patient, had progressed uninfluenced by any treatment.

(4) Herpes zoster corneae, or its complete manifestation, herpes zoster ophthalmicus, is an epidemic infection affecting primarily nerve tissue. As a rule, it is unilateral. An attack usually confers immunity. The disease begins as follows: there is neuralgia and pain along the course of the fifth nerve, which precedes or occurs simultaneously with the second or eruptive stage. Fever may be present and flushing of the skin. Vesicles appear, becoming purulent. After healing, scars are left behind. Ocular symptoms develop early. The cornea becomes involved in at least 50 per cent of the cases. Diminished sensibility of the cornea is present; an ulcerative keratitis may be destructive to the eye. Iritis is often present. I have seen striations in Descemet's membrane, suggesting hypotension. Recovery takes place in two to six weeks. Treatment still remains symptomatic.

Gruter[20] gave an impetus to the study of the virus infection by discovering the transmissibility of the herpes virus and the close relationship of the various herpetic forms. Following his lead, Levaditi and his pupils in the Pasteur Institute and Goodpasture and Teague[21] in this country have, by careful scientific research, given the profession some valuable information concerning the mode of action and the varying toxicity of the herpetic virus.

They have shown by repeated experiments that the virus passes from the periphery (cornea, mucous membrane, or skin) along motor, sensory and sympathetic nerves. Goodpasture and Teague believe the transmission is along the axis cylinder of the nerve. Others believe that the infection travels along the perineural lymph sheath. Experiments have shown that the virus

can assume various degrees of virulence. Under ordinary circumstances the herpetic manifestation is peripheral; for example, herpes corneae, or herpes libialis. Passing the same virus through a series of rabbits may increase its virulence. Inoculation of the corneae of rabbits with the more potent virus produces herpetic encephalitis. Goodpasture and Teague think that simple herpes and herpes zoster result from the same virus, differing only in virulence. They took a virulent strain of the simple herpetic virus, inoculated rabbits' corneae and produced a serious encephalitis. Then, taking the virus from the encephalitic rabbits and inoculating the skin of guinea pigs, they were able to produce in guinea pigs a zonular eruption resembling herpes zoster. As in zoster, it was found that the posterior root ganglia had been affected.

However, Lowenstien's[22] experiments convinced him that the viruses are not identical. Other authorities are equally certain that the two diseases are different. They regard zoster as a true exanthema, closely related to chicken pox. Epidemics of the two diseases occurring simultaneously have been reported. Zoster and chicken pox confer an immunity. Some have seen an interrelationship between the immunity conferred. Paton,[23] in a masterful discussion, gives his reason for believing chicken pox and herpes zoster are kindred virus infections.

The primary lesion in herpes zoster corneae is in the Gasserian ganglion. The infection gains entrance through the mucous membrane of the mouth or nasopharynx and is conducted to the ganglion along the nerve sheath or directly through the axis cylinder of the nerve.

Treatment.—Simple herpes requires no special treatment, except protection. A confluent lesion involving a large area of corneal surface should be treated by rest in bed. In addition to a protective dressing, the lids should be closed daily by adhesive tape. Atropin is indicated. A sluggish herpetic lesion showing no tendency to heal will often respond if its edges are carefully curetted, which destroys all the unhealthy epithelium. The usual protective dressing should be used. This treatment has likewise been used in recurrent erosions and successful results have been reported. According to Duke Elder, ultraviolet light therapy has given beneficial results. The treatment of herpes zoster remains symptomatic, yet great activity is now going on toward developing a curative serum.

(5) Neuropathic or neuroparalytic keratitis results from diseases, injuries or operations affecting the Gasserian ganglion or branches of the fifth nerve. Oculists for the most part see these cases following operations or injections for the relief of trifacial neuralgia.

Conditions leading to keratitis are: infrequent winking and reduction in the secretion of tears; anesthesia of the cornea. Symptoms of keratitis are: dullness and dimpling of the corneal surface; exfoliation of the epithelium; infection, followed by rapid destruction of the tissues. Not all patients operated upon or injected develop neuropathic keratitis. The reason some cases do suffer has never been definitely settled.

(1) The trophic theory is based on the hypothesis that certain nerves from the sympathetic control the nutrition of the cornea. (2) The desiccation theory that it is due to infrequent winking fulfills some conditions. (3) The traumatic theory is based on the fact that injury occurs to the cornea because of its anesthesia. (4) Verhoef[24] advances a plausible theory, to say the least; his theory is that there is diminished lacrimal secretion. He believes that the lacrimal gland is supplied by secretory nerves from both the fifth and the seventh. It is only in those cases in which the secretory fibres from both nerves are injured or destroyed that neuropathic keratitis results. Injury can easily happen to the branches of the facial within the great petrosal nerve as it passes under the Gasserian ganglion.

Treatment.—Verhoef cured a case by keeping the cornea constantly moistened. Later, Ringer's solution was instilled in the eye every two hours. The safest and surest treatment in all cases threatened is to keep the lids closed by a permanent tarsorrhaphy until the danger has passed.

III.—CORNEAL DYSTROPHIES

Corneal dystrophies show a number of variations. Some forms of them are frequently seen in ophthalmic practice. A dystrophy is a condition in which body tissues and tissue fluids are no longer able to dissolve and carry away the products of metabolism in a given structure. Fuchs says that dystrophies are characterized by absence of inflammatory reaction and invasion of leukocytes; by a progressive course; by fatty, hyaline and calcareous degeneration. Dystrophies fall into two classes: primary and secondary. The secondary forms are usually zonular and bullous keratitis. These two will be discussed later.

Primary dystrophies are divided into these classes: (1) familial, or hereditary; (2) occupational; (3) essential; (4) senile.

The familial types begin about puberty, are bilateral and affect one or more members of the same family. Marcus Gunn[25] reports four

cases in a family of ten. Dunbar Roy[26] records six cases in one family, mother and five children. Collins[27] gives a pedigree chart showing four generations affected. He refers to Spicer's report of observing the disease in three generations. The course is progressive. Familial or hereditary dystrophies assume various forms and shapes: nodular (Groenouw); reticular, or lattice-like; punctate; flake-like and disc-like opacities. Authorities now regard all these as different manifestations of the same affection. It has not yet been determined whether the condition results from a neuritic disturbance or is due to a primary degeneration of the corneal stroma. The location in the superficial stroma and the slight decrease in the corneal sensibility inclines one toward the neuropathic theory.

Fuchs,[28] describing cases of nodular keratitis seen by him, says:

"The opacities occupy the center of the cornea; there is gross unevenness of the corneal surface; grey spots centrally located often project above the corneal surface; smaller spots surround the larger ones in a circular manner; the opacities are round or irregular; microscopically, a substance is seen deposited beneath the epithelium and in the superficial stroma."

Uhthoff[29] gives a histological report of one case. There was nodular thickening of the deeper layers of the epithelium. The nodules lay in pits in the superficial stroma. Hyaline degeneration of the superficial layers of the cornea was noticed. Chou[30] examined a case with the corneal microscope. Opacities were discs, nodules, and flakes. The corneal nerves were thickened and visibility to the smallest branches was observed. Byer[31] reports a case of reticular dystrophy. The opacities as seen by the corneal microscope showed a net-work of grey lines beneath Bowman's membrane. A recent article by Fuchs[32] calls attention to the visibility of the nerves to the naked eye in this form of dystrophy. Keif,[33] basing his opinion on his own experience and that of others, concludes that no treatment, including prolonged light therapy, influences the course and progress of familial dystrophies in the slightest.

Two cases of unusual corneal dystrophy have been seen by me during the past two years.

The lesions are similar in the two patients; they are bilateral and appear as grey-white irregular central opacities. One case is in a young woman, age 42. She has a low error of refraction. The second case is in a woman of 65. Both women are in good health. Physical examinations, blood chemistry and other examinations fail to reveal a cause. The older woman states that the reduction of vision has been slow and gradual; it is reduced to 4/200. The younger woman still has fair vision. The opacities seen by the corneal microscope are fine dots grouped together, more dense in the center. The slit light shows the dots are in the outer stroma, but on different levels. There is no increased visibility of the corneal nerve filaments. Neither case gave any history of ocular inflammation.

Black and Haesler[34] report a case of peculiar dystrophy limited to the epithelium alone, causing no thickening of the layers. They regard the opacities as being intra-cellular protoplasmic changes. They are unable to state whether it is due to fatty or other substances. The fact that it was relieved by thermaphore treatment argues against its being a true dystrophy. Meyer[35] described the case of fatty dystrophy (dystrophy adiposa) centrally located in the corneae of both eyes, occurring in a woman sixty-five years old, due to a degenerative fat infiltration. In this case the blood serum ran a high lipoid content. He regards it as a true dystrophy of endogenous origin. Chance[36] reported a similar fatty dystrophy. Result of physical examination or blood chemistry was not given.

Dystrophia Epithelialis Corneae.—This epithelial dystrophy of the cornea, first described by Fuchs, is usually bilateral and occurs mostly in women past middle life. In these cases there is a decreased sensitiveness of the cornea. The surface is uneven and dull. One to several central epithelial vesicles are seen, associated with dark depressions where vesicles have ruptured. The course is slowly progressive. Early observers thought the changes were only in the epithelium and superficial stroma, but the corneal microscope shows endothelial changes as well. Often changes are noted in the endothelium first. This fact has been commented on by Friedenwald.[37] Gifford[38] thinks that recurrent erosions of the cornea and vesicular keratitis are forms of epithelial dystrophy. Fuchs[39] regards this dystrophy as primarily a disease of the corneal nerves.

In 1924 and 1925 I had under observation a classical case of this disease. A woman, age 65, was in good health. She had a bilateral dystrophy of five years' duration when seen by me. Vision of each eye was less than 20/200. A search failed to reveal a cause. Treatment had no effect in the course of the disease. Heat from the thermaphore seemed to bring on recurrences. A recent report from the patient states that she is now almost blind.

The case reported by Langdon[40] as a true epithelial dystrophy, cured by a diet rich in vitamin A in the form of cod liver oil and butter, must, in my mind, be regarded as a form of nutritional keratitis. I have been unable to find the report of a single case of true dystrophy of this kind cured by either diet or treatment.

Arcus senilis is probably the purest type of the so-called senile dystrophy. It certainly is the most common. In fact, it is so frequent that by some text books it is called physiological. It is mostly the rule in old age. It occurs often in middle life and is sometimes seen in youth,

due, Fuchs says, to early degeneration or due to continuous action of external irritants. Consensus of opinion is that it is a form of fatty degeneration. According to Munson, it is due to an increase of cholesterin and other deposits in the blood. Meyer[41] studied the beginning of gerontoxon in 108 cases between the ages of five and seventy-five years with the corneal microscope. He found that the condition begins between the ages of fifteen and thirty years, showing first peripheral opacities in Descemet's membrane. Later, peripheral opacities come in Bowman's membrane. Very gradually the opacities are seen to join.

Closely allied to arcus senilis is marginal dystrophy of the cornea. It is not an uncommon observation to see this marginal furrowing of the cornea in people well advanced in life, involving only a small area, but more commonly the entire circumference of the cornea. Yet, good vision obtains. A recent case illustrates my point.

A man, age 84, always healthy, had never suffered from any inflammation of either eye. He had no visual disturbance. Corrected vision was 20/20. Each cornea showed a complete deep circular groove just inside the limbal margin. The scleral edge assumed a gradual slope. The central ledge was a distinct grey-white opaque ring which was abrupt and steep. A marginal network of small vessels could be seen traversing the bottom of the groove. Perhaps the final stage of ectasia would result in all these cases if they lived long enough.

While we regard such cases as senile degeneration, there is no doubt that a marginal dystrophy can also result from a low-grade inflammatory process. Reports of such cases are too convincing to ignore. The disease begins often in early life, progressing slowly but continuously to the final stage of ectasia.

Gonzales[42] reports a case in which the disease was first noted at the age of 16. He was able to observe over a period of ten years the slow progressive thinning to its final ectasic stage. S. R. Gifford[43] gives a detailed account of three cases, ages 29, 33 and 32 years. All showed recurring inflammatory attacks during the course of the disease. Doggart,[44] in a very interesting article, reports a case beginning at the age of thirty and ending after a long period of years in ectasic marginal dystrophy. The patient had many recurrences of inflammation.

Treatment with the use of the cautery, and in some instances, the cautery and the free use of the conjunctival flap together, have given favorable results in the improvement of vision by reducing the astigmatic error created by the ectasia.

Zonular keratitis occurs both as a primary and a secondary dystrophy. The secondary form is seen fairly often. It results from a perverted nutrition of the eye itself. It occurs in eyes blind from glaucoma, iridocyclitis and injury. The primary form results from mechanical thermic and chemical irritants and may be classified as occupational. Due to the irritation of flying particles of hair numerous cases have been reported in people who are employed as hatmakers. It is said to occur in those who are exposed to mercurial vapors. The changes are characterized by a deposit of sclerotic tissue beneath Bowman's membrane, destruction of the superficial stroma with finally hyaline and calcareous degeneration. Zonular keratitis usually begins at the limbus and extends across the lower part of the cornea as a grey rough opacity several mm. in width. Appleman[45] reports a case in a hatter, improved by treatment. Merkulow[46] reports improvement of a child treated with x-ray.

Keratitis bullosa is a type of secondary dystrophy which is often seen in eyes long under increased tension or otherwise diseased. The walls of the bullae are a form of connective tissue. This disease represents a sort of transitional stage which ends in sclerosis of the cornea (Meller). Recurring bullae always excite considerable pain and discomfort.

In stopping recurrences no treatment is more effective than tearing away the bulla and curetting the unhealthy or diseased epithelium around the edges of the bullous area. Primary vesicular keratitis is allied to neurotrophic disturbances and is discussed under that heading.

Kruckenberg's Spindle.—Sufficient data have now been collected to classify this peculiar corneal condition as a dystrophy. It occurs as a spindle shaped, brown-red opacity in the center of the cornea. The opacity is deep in the corneal stroma, due to punctate opacities grouped in a spindle shape. It is not congenital; it is bilateral and symmetrical. No antecedent inflammation has been seen in the cases reported. It usually occurs in myopia. The youngest case in which the opacities were seen was 22 years old.

Kruckenberg reported 3 cases in 1899. Thompson and Ballentyne[47] report one case. Friedman[48] reports one case. Edgerton[49] gives a resume of cases reported.

In this paper I report another case, Mr. B., first seen in 1926; healthy, but a nervous man. He has bilateral myopia of 10 diopters. Corrected vision is 20/30. The typical spindle is plainly seen. The color is characteristic. The slit light shows the opacities to be punctate and located in the deeper layers of the cornea and in Descemet's membrane. The opacities are

more dense in the center. There is no antecedent inflammation of the eyes. For four years the condition has been stationary. Careful examination failed to reveal a cause except the myopia.

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DISCUSSION (*Abstract*)

Dr. Kate S. Zerfoss, Nashville, Tenn.—After tuberculous treatment in a group of cases with phlyctenular keratitis it was observed that there was a prompt improvement in the subjective symptoms. The pathological changes diminished, to be followed by an exacerbation of both later. Still later there appeared to be a definite and constant improvement. The x-ray of the chests of these individuals was negative except for a healed hilus tuberculosis. The large number of young adults in this group was striking.

As improvement is found in the handling of trachoma cases, as indicated by the essayist, we should certainly follow the routine outlined of hospitalization, and so forth. It is amazing that so far there has been no clinical correlation with the findings of Noguchi in this disease.

An excellent summary of the eye manifestations of vitamin A deficiency has been given by Wolback and Howe. It is as follows: (1) a keratinization of the corneal epithelium; (2) edema and vascularization of the parenchyma of the cornea; (3) leukocytic invasion; (4) an encrustation of the lids due to the desquamated cells. They observed a few cases of pyogenic infection.

The essential change is the tendency for the epithelium in the body to be converted into a keratinizing type. It is found in the cornea, lacrimal glands and also in the respiratory, intestinal and genito-urinary systems. The rat has been used in experimental work and found to have this transition. However, when the guinea pig was subjected to the diet without vitamin A (as described by Osborne and Mendel) it was found that the corneal epithelium was not affected, while there was a generalized keratinization of the epithelium elsewhere in the body. In all cases the change of the type preceded the atrophy of the cell.

Another point of significance, aside from the species difference, in the development of xerophthalmia, was that the rats which had been castrated were found to develop the ophthalmic changes from three days to three weeks later. This observation was made by Mason. His suggested explanation was that perhaps the altered metabolic rate influenced the delay.

In studying the herpetic group of keratitis we note that the simplex type may be and often is recurrent, whereas herpes zoster of the cornea is practically always characterized by a single attack.

Goodpasture and Teague regard the etiological factor, herpetic virus, as the same in each instance, the difference in the manifestation being due to the virulence of the virus. The immunity conferred is lasting in herpes zoster and may be temporary in herpes simplex. They indicate also that the virus is transmitted by a proliferative process in the axis cylinder.

One is constantly impressed with the incidence of herpes of the cornea occurring with acute respiratory infection; also focal infection. The depth of the lesion in the cornea can be well identified with the corneal microscope. The extremes of this, from the standpoint of depth, vary from the superficial, vesicular type to the deep involvement of the parenchyma in herpes zoster. An intermediate level of pathological change occurs below the epithelium. Keratitis punctata superficialis shows no tendency to form vesicles or to become ulcerated as do the others. In one case of herpes zoster observed there was an accompanying iritis and a rapid development of a lenticular opacity. It is well to give an early conservative prognosis in such cases. The destruction of the cornea from keratitis neuroparalytica in the severe form seems to be the same, whether the nerve was sectioned or an alcohol injection was made in the Gasserian ganglion.

Again, the use of the corneal microscope enables us to obtain a more complete and accurate study of the exact location of the lesions in the corneal dystrophies.

The calcareous deposits beneath the epithelium seem to be the one type that offers improvement by treatment. In the case reported by Appleman the film of calcium was removed by scraping, after the overlying epithelium had been removed. The group incorporated in the occupational dystrophies can be prevented if attacked at the source by the provision of safety measures.

Vogt's observation of arcus senilis indicates that there is often an extension of the altered corneal tissue in the deeper portions to the corneal-scleral junction. Vogt regards the fusiform pigmentation of the posterior surface of the cornea as being nearly always bilateral and occurring in cases of myopia only. Considering the other degenerative changes that appear in the cases with a high degree of myopia, it seems plausible to think of Kruckenberg's spindle as coming into this category.

Dr. Meyer Wiener, St. Louis, Mo.—The author has presented a classic on the subject of keratitis. The arrangement of types is given in a logical sequence, with a sensible grouping of the different forms of corneal involvement. While we do not wholly agree with all of his theories, we must admit that he advances some splendid reasons in presenting his various expressed thoughts.

Phlyctenular disease is undoubtedly far more frequent in New Orleans than we find it in St. Louis, either in

clinic or private practice. Personally, I am inclined to lean toward the tuberculous theory of phlyctenular disease. There is no question that diet and sunshine have a tremendous influence on the progress of this disease, but they would on tuberculosis as well. Dr. Buchanan's observations on the clinical treatment of trachomatous pannus, bearing out Stucky's contention that it is a nutritional deficiency disease, are most interesting and deserve further observation and confirmative investigation. The case of Mrs. H., who complained of a dry and scratchy feeling, and was finally relieved by a diet rich in vitamins A and B, forces us to the conclusion that we may be failing to solve some of our seemingly simple, but really obscure and stubborn, cases of conjunctival irritation.

I have seen and reported superficial punctate keratitis with no impairment of vision, no redness or irritation, the only complaint being that of tearing. Recurrences occurred at intervals within a six-year period, no cause ever having been found. Here the lesions were seen only with the aid of the loup and were discernible chiefly as minute round, raised epithelial areas scattered over the entire cornea. They eventually disappeared.

I was taught by my preceptor, Dr. Charles Michel, that herpetic or dendritic keratitis was always malarial, but soon learned that many other factors had to be considered, chief among them being focal infection.

Keratitis following operation on the Gasserian ganglion can be avoided by closure of the lids and frequent irrigation with non-irritating solutions. If your patient is not intelligent, tarsorrhaphy is the safest procedure.

I agree with the author that there is no known treatment which has any influence on epithelial dystrophy. A few years ago Professor Fuchs sent me a case, a gentleman from North Carolina, who had consulted him in Philadelphia. He thought the condition, being superficial, might be benefited by a resection of the entire corneal surface. About one-half of the thickness of the entire cornea was removed. It was epithelized in about ten days and much clearer than before operation, with improved vision. We were doomed to disappointment, however, as far as the ultimate outcome was concerned. The same defect in metabolism which caused the original condition produced a thickening of the epithelium and sub-epithelial tissues, beginning at the corneal margin and slowly progressing toward the center. I have tried this twice, both times unsuccessfully.

Upon the recommendation of Dr. Richard Weiss, a dermatologist, we administered sodium iodide intravenously in 30 grain doses to a patient with severe keratitis bullosa. An immediate and continuous improvement was noted.

Dr. J. A. Stucky, Lexington, Ky.—I agree with the conclusions of the essayist regarding the etiology of the different forms of keratitis. I have not found the number of cases of phlyctenulosis and pannus among the negroes which he mentions. Indeed, I have seldom seen a case. The disease, as he said, is essentially and directly the result of a basic disturbance of nutrition, worse in those with a constitutional predisposition.

In my work the treatment is divided into (1) the surgical, (2) local and medicinal, (3) nutritional. If we add to the latter a high vitamin A diet with sunshine and fresh air, I have found the sordid surroundings of the patient will not be so important a factor in the ultimate cure. In addition to this I give at least two doses of pure cod liver oil daily for several months after hospitalization. Usually the latter does not last over thirty days.

I am convinced that where vitamin A is needed B and D are also indicated, though not to the same degree. I firmly believe in the value of synergistic nutritional as well as remedial treatment. In the majority of instances Old Mammy Nature does not fire a rifle, but a shotgun.

The most important thing to bear in mind in the treatment of these cases is that, since they are the result of a nutritional deficiency, lack of vitamin A, sunshine and fresh air, all of these must be continued indefinitely.

In many cases it is as difficult to correct the nutritional disaster as it is to correct the condition for which the patients consult us.

There is yet as much to learn about nutrition and balanced feeding as there is about allergy and immunity. The trouble with most of us is that we have been living in a rapid, spectacular age for so long that we are loathe to go back to the methods of restoring and rebuilding the body deficiencies through the slow process of nutrition and cellular activity. It is unfortunate, too, that dietetics and nutrition are treated as if beneath the dignity of the specialist. We say in general terms, take a nutritious diet, a light diet, a soft diet, but how many of us know the meaning of these? How many of us can explain to the nurse and patients how, what and when we want our patients to eat?

Dr. Richard S. Weiss, St. Louis, Mo.—I rise to confirm what Dr. Wiener has just said about the patient with keratitis bullosa. Dr. E. Wood Ruggles, about one year ago, reported a series of cases of herpes zoster, including cases in which the ophthalmic division of the fifth nerve was affected, that he treated with intravenous sodium iodide injections with remarkable results. We used sodium iodide in one case and our patient got perfectly well.

Sodium iodide is administered intravenously in 2 gram doses; the second dose on the third day; the third dose on the fifth day, and the fourth dose on the eighth or ninth day. In this type of ophthalmic case it would be advisable to continue the injections for fifteen doses if necessary before discarding the treatment.

Dr. John G. McLaurin, Dallas, Tex.—I am very sorry that Dr. Buffington did not have an opportunity to go into the details of the treatment for phlyctenular conjunctivitis and trachoma. He referred to a case of keratitis bullosa which I had asked him to see as a consultant some time ago. This patient was a woman who had recurrences of keratitis bullosa at least 25 times in one eye while she was under my care and she gave a history of having had the same experience with the other eye before I had seen her. Dr. Ernest Fuchs, of Vienna, who saw her, made a diagnosis of keratitis vesiculosa externa. The patient was in her menopause and I am inclined to think that this type of trouble may, in some cases, be influenced by endocrine imbalance as well as nutritional disturbances. The patient's nutrition may be upset at that time of life on account of the menopause. After trying various forms of medication and measures designed for the removal of the bullae and a regrowth of epithelium that would be firmly attached, I finally stopped all medication and, under proper regulation of her diet and by keeping the eye covered without local treatments,

the patient completely recovered. This plan of treatment was in accordance with Dr. Fuchs' suggestions.

There is another type of keratitis bullosa that is sometimes seen that might be quite troublesome, the so-called traumatic recurrent keratitis bullosa. It is more likely to follow operative procedures upon the eye.

There are two other types of keratitis that can be easily confused. One is the herpetiform type of keratitis following febrile disturbances, ordinarily known as herpes febrilis; the other, the so-called keratitis punctata superficialis. They can, however, be easily differentiated. The punctata type does not often have vesicle formation and the little grey beads formed on the cornea are readily recognized with the loupe after one has seen such a case. Ten to 100 of these small beads may be seen on the cornea. They do not appear for two or three weeks after the recovery from the disease that has been responsible for them.

Dr. William Thornwall Davis, Washington, D. C.—Phlyctenular conjunctivitis may be divided into two forms: one is a type due to metabolic or nutritional disorders. As a rule that is a mild form and responds to dietary and hygienic treatment rather promptly. The other is a more severe and serious type which does not respond to those measures, and which invades the cornea, travels across the cornea by progressive ulceration, and finally not only interferes with vision, but perhaps destroys the eye. It seems to me that that form, clinically at least, may be separated from the less severe form. The more severe form is probably due to a tuberculous lesion, because it is in the severe form that we get a positive tuberculin reaction, whereas in the less severe form, due to nutritional disturbance, we do not get a positive reaction.

These two types separate themselves rather clearly in clinical work, and if you observe them you will see that at least 95 per cent will dissolve themselves rather promptly under nutritional measures, whereas the other form will not be affected.

BENACOL IN RECTAL CONDITIONS: EMPHASIZING ITS USE IN RECTAL SURGERY*

By HERBERT T. HAYES, M.D., F.A.C.S.,
Houston, Texas.

I believe that in the foregoing years there has been more undue or unnecessary trauma during rectal operations than in any other form of surgery. As a rule, this results in the patient's having a great deal of pain; and consequently, in the lay mind, all rectal operations are associated with pain and suffering. I think perhaps a greater number of people have refused operative procedures from this one cause than from any other. Needless to say, there are some operations which will always give pain. Elimination of pathological rectal conditions with good end-results is the paramount consideration in rectal surgery, but another thing that we should strive for, in order to have a satisfied patient and one who will be glad to refer more work, is to keep our operative cases as comfortable as possible.

Stone, in 1926, advocated the use of alcohol injections around the anus after hemorrhoidectomies for the relief of pain. I know of other men who have also been using alcohol with good results. A weak solution of quinine and urea has been recommended by others. Both of these drugs give anesthesia for a number of days. I have used alcohol for temporary relief in pruritus, but have never used it following hemorrhoidectomies. I have also used quinine and urea, but think healing is delayed too much following its use.

In 1927, Yeomans and his associates recommended the use of benacol in the treatment of pruritus, and at a later date recommended its use in the treatment of fissure in ano. Yeomans states:

"Benacol solution consists of equal parts of para amino benzoyl ethanol benzoate and phenmethylol in ninety parts of rectified sweet almond oil. The chemical formula is $C_{16}H_{17}O_4$ N and C_7H_8O. It is nontoxic, highly anesthetic, moderately antiseptic and, as far as investigated, has a mild depressant action on peripheral nerve endings."

I began using benacol injections for pruritus in 1927, and had used it in 14 cases by 1929. In some cases the results were very good, but in others not so satisfactory. The fact that caught my attention was the persistent anesthesia following the injections. This would often last a week and sometimes two weeks or more. Many of these cases, as you know, have multiple fissures and suffer from aching and burning pain; this was, of course, relieved. In 1929, I began using benacol to inject under the painful fissures of a more extensive nature that are seen in the anterior and posterior commisures. The results were so gratifying that they led me to begin using it in nearly all of my rectal surgery. Yeomans stated that it was antiseptic and anesthetic, and I have found that with these two qualities, and unlike alcohol or quinine, it does not delay healing. However, if a large amount is puddled in one spot, there will be an indurated area for some time and a slight delay in healing.

In some cases, the pain is entirely eliminated and in others, while there is a little pain, it is greatly alleviated. The effects of the drug last for a week or more and after-treatment can be

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given with much more comfort. Another result in favor of this procedure is that the patient is able to get out of bed and about his business at an earlier date. We do all hemorrhoidectomies and extensive fistulectomies in the hospital, but I have been tempted to do more of the hemorrhoid operations in the office, and would, were it not for the time consumed in making house calls afterward. All of the minor cases, such as thrombotic hemorrhoids, fissures and straight-tract fistulas, can be done in the office with no detention of the patients from their work, and with no discomfort or danger of having to catheterize the patient.

In all cases the incidence of catheterization is decreased, because the pain and sphincteric spasm are reduced. The relaxation of the sphincter facilitates the drainage from the raw areas and lessens the danger of infection. There is theoretically some danger of infection from injecting any substance about the rectum, but when the proper technic is followed this danger is very slight. I have used it on a total of 212 cases with an infection in only one case.

The following is a summary of the cases in which benacol was used, with an analysis of these cases and the technic of the injection. The surgical cases are all from my private practice and were operated upon during the past twelve months:

In 55 hemorrhoidectomies in which benacol was injected, many patients had no pain whatever; and there was a marked lessening of the pain in all but three or four patients, who had considerable discomfort which could not be explained. If a local anesthetic is used, this is given in the usual way and then the benacol is injected. To inject benacol, the needle is inserted on either side of the rectum, close to the anal margin, and the solution is distributed along one side of the anus through this single puncture. The operation is then performed. I believe that to inject the solution after the operation makes the patient liable to infection.

External thrombotic hemorrhoids are anesthetized with novocain and a small amount of benacol is injected under the areas before they are excised. In fistulectomies, the benacol is injected under the areas involved after the anesthetic is administered. Anal crypts and papillae are anesthetized in the usual way and a few drops of benacol injected under each area. In excising or incising small abscesses, the benacol is injected under the area. Small fissures that have no capping and overhanging polyp or papillae usually heal after injecting benacol under them. It can also be used in the more severe fissures or ulcers to give temporary relief until they can be operated upon.

I have injected benacol under thrombotic hemorrhoids with and without novocain and then have done nothing else to them, except apply hot compresses and allow them to shrink. The injection lessens the pain, relaxes the muscle, and thus relieves the congestion, allowing the area to shrink more rapidly. This mode of treatment is particularly applicable in women before and immediately after childbirth, and in quite a number of other cases where excision is not deemed advisable. It is best to give all these minor cases a grain of codein and ten grains of aspirin; this will control the burning that occurs after the novocain wears off. After hemorrhoidectomies, one hypodermic of morphine is usually sufficient to take care of the discomfort that begins as the anesthetic dies out.

SUMMARY OF CASES IN WHICH BENACOL WAS USED

Operation	Number	Catheterized	Not Catheterized	Anesthesia		
				Local	Spinal	General
Hemorrhoidectomy	55	20	35	36	12	7
Excision of Thrombotic Hemorrhoid	25	0	25	25	0	0
Fistulectomy	26	3	23	17	9	0
Excision of Fissure	27	2	25	26	1	0
Excision of Anal Crypts and Papillae	7	0	7	7	0	0
Excision and Incision of Abscess	5	0	5	4	1	0
Fissures Injected	25	0	25	25	0	0
External Hemorrhoids Injected	11	0	11	6	0	0
Pruritus Ani Injected	31	0	31	31	0	0
Total	212	25	187	177	23	7

In the 52 fissures, 27 were operated upon in the usual way, after they were infiltrated with novocain and benacol was injected under the operative field and into the muscle on each side. Benacol was used in 31 cases of pruritus, with considerable benefit in some cases and always some temporary relief.

CONCLUSION

After using benacol on 212 various rectal conditions, I believe it is a valuable aid in relieving pain in surgical cases and is of benefit in the treatment of pruritus.

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Discussion follows paper of Dr. Durham, page 312.

GASTROINTESTINAL SYMPTOMS IN ANORECTAL DISEASES*

By F. M. DURHAM, M.D.,
Columbia, S. C.

Dr. Oliver Wendell Holmes, in discussing the sphincter ani before his classes in anatomy, used to say:

"This muscle is very small and its function is menial, but I tell you, young gentlemen, upon its integrity rests the very foundation of society."

I do not wish to create the impression that most of the gastrointestinal disturbances have their beginning in anorectal diseases, but, on the contrary, I sincerely believe that more rectal disturbances have their etiology in diseases higher up in the gastrointestinal tract than are produced from the anorectum upward. I believe that about four-fifths of the causes of rectal disturbances come from points higher up in the gastrointestinal tract, and that about one-fourth of the cases of gastrointestinal disturbances are influenced from the anus and rectum to points higher up in the gastrointestinal tract, and that the anorectal influence on gastric symptoms is, as a rule, overlooked in the general examination.

Just why the rectum has been neglected or overlooked as a cause of gastric disturbance has always remained a mystery to me; for even in intractable gastric symptoms, where no cause can be found in the stomach, every physician, even the most careless, would certainly eliminate cholecystitis and appendicitis, and know the general stability of the nervous system of the patient before he would attempt a diagnosis. Every patient suffering from an intractable digestive disturbance usually gets a thorough physical examination of every portion of the body with the exception of the rectum.

Many a case of so-called nervous indigestion which has driven the sufferer to despair, causing him to go from one physician to another, and whose digestive trouble has been looked upon as a "case of nerves" has its etiology in an anal ulcer or an internal hemorrhoid. Nervous indigestion, gas, heartburn, belching and backache may be the results of an internal hemorrhoid or a proctitis. Nervous indigestion, undue fatigue, anemia and mental depression may be associated with a small internal hemorrhoid that loses a small amount of blood at each bowel movement.

An irritable sphincter ani can set up a train of digestive disturbances that will simulate a patulous appendix, or a stone in the common duct. An hypertrophied sphincter ani is a prolific cause of constipation and though the constipation produces every digestive disturbance that a poor human being is capable of having, purgatives do not seem to remove, even temporarily, the auto-intoxication in such cases as they do in most other cases of constipation. Dyschezia is supposed to be caused by a form of constipation that is locally confined to the rectum and is often responsible for a train of digestive disturbances and symptoms of auto-intoxication.

I have seen patients gain in weight and become relieved of constipation when an anorectal polyp was removed. I have seen patients very much depressed, nervous, below normal weight, constipated and with no appetite, take on weight, be relieved of constipation and become cheerful after the removal of an infected anal crypt.

Many nervous, anemic women with a train of digestive disturbances and with a history of menstrual disorder and bladder symptoms have been relieved by the removal of large, hypertrophied papillae, or the curing of an anal ulcer.

I often hear my stomach patients say their digestion was worse when they had a "little touch of the piles." I have heard many patients state that they no longer suffered from "stomach trouble" since their piles had been injected or operated upon.

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Many patients have told me that, since their anal fissure had been cured, they were no longer constipated, their appetite was good and they were sleeping well. I have seen a patient gain twenty pounds after relief from anal fissure. I have seen gastric and duodenal ulcer patients have decided exacerbation following an acute attack of hemorrhoids. I believe that many of the temporary spasms and regurgitations following gastroenterostomies are caused by acute conditions in the anus and rectum.

Many cases of acute gastritis are preceded by an acute exacerbation of hemorrhoids. Every few days, I hear patients make the remark that they are not nearly so nervous, that they sleep better, that their digestion is improved and their constipation is relieved since they have been cured of an anorectal fistula, although the fistula may have been small.

With the great progress that ambulant proctology has made in the development of simple and painless methods by which these minor anorectal conditions can be relieved in the physician's office without hospitalization, there is no excuse for the gastroenterologist not to use his proctoscope. There is a great future for gastroenterology if the gastroenterologist will not overlook his proctology.

I will not be quite so extreme as to the importance of some portions of the anatomy of the gastrointestinal tract as was Dr. Oliver Wendell Holmes, but I will say that one person out of every six in the civilized world suffers from some anorectal disease and that it will pay the gastroenterologist to watch the anus and rectum in all of his intractable and nervous stomach cases.

DISCUSSION (*Abstract*)

Papers of Dr. Hayes and Dr. Durham.

Dr. Walter C. Alvarez, Rochester, Minn.—There is no question that there are some patients who can be cured of constipation and flatulence by treatment directed toward the healing of infected fissures about the anus. I have had a number of very happy patients who were cured in this way, but in many others removal of even large irritated hemorrhoids did not relieve the attendant constipation. There seems to be some relation between irritable hemorrhoids and the presence of gas in the splenic flexure.

It is unfortunate that few physicians examine the rectum. Most of the carcinomas of the colon are within easy reach of the examining finger, and yet in most cases the diagnosis is made so late that the surgeon cannot remove the lesion. Even when hemorrhoids are present to account for the bleeding complained of, there may be a carcinoma three inches farther up.

Dr. Granville S. Hanes, Louisville, Ky.—I have had a very limited experience in the employment of benacol in the treatment of rectal diseases. After it was recommended highly by Dr. Yeomans for the relief of pruritus ani I treated a limited number of patients, but did not obtain the good results I anticipated.

Since I have treated many pruritic patients with hydrochloric acid, with most satisfactory results, it is quite likely that an element of prejudice entered into my estimation of the relative value of the two agents.

I injected hydrochloric acid to relieve this tormenting and intractible trouble, because I had become convinced that the cutaneous tissues were not primarily affected, but that the tissues under the skin harbored the real cause. Numerous agents were injected under the itching surfaces, but with results that were by no means satisfactory. Hydrochloric acid produces an active reaction when introduced into the tissues; it is germicidal and it is a natural secretion of the stomach. On these empirical grounds I experimented with hydrochloric acid and found it by far the most reliable and potent agent I had used. It is inexpensive and can be employed in extensive areas. Of course, its introduction must be preceded by injecting about twice the amount of 1 per cent novocaine, as the hydrochloric would otherwise produce intolerable pain. Until one has become familiar with its use it would be well to treat limited areas, using small amounts, and thus note the results. Where the tissues are very loose and fluids distribute into the surrounding tissues easily, there is not likely to be much pain or subsequent breaking down of the tissues.

After we had used hydrochloric acid for two or three years we began to appreciate the value of vigorous massage of the parts after treatment. The parts should be massaged and pounded after treatment for a half-hour to an hour and this treatment should be repeated three or four times a day until the soreness entirely disappears. Massage is of itself stimulating and is an excellent treatment for pruritus.

We are convinced that the original source of pruritus ani, vulvae and all similar types of itching about the genitals comes from bacteria and toxins in the bowel. Medication must, therefore, be instituted, both by mouth and the lower bowel to obtain permanent relief. When I say medication, of course, I include diet, which is very important.

I have never seen any one severely affected with a typical pruritus ani who could not be relieved at once by the injection of hydrochloric acid. If, however, a permanent cure is to be effected the primary cause, which is in the bowel, must be relieved.

For two years we have been injecting sterilized castor oil, added to which is 0.5 per cent phenol and 0.5 per cent nupercaine. Of course, this injection is preceded by novocaine as in hydrochloric injections. The oil is used in about the same amounts as the acid and it may be employed in at least 80 per cent of pruritic cases successfully. In the most intractible cases of itching we depend upon the acid.

Hydrochloric acid has more than a local influence when injected into the tissues. It is a most effective agent when injected under eczematous lesions on the extremities and other parts of the body. Many times we have observed that when it is injected under an eczematous area on the arm or leg, a similar lesion on the opposite extremity is relieved just as promptly and effectively.

Hydrochloric used for injections is prepared from the pure acid in the strength of 1-3000. The injection of the acid may be repeated in from one to two or three weeks. This depends upon the reaction and relief obtained from each treatment. Aspirin, codeine and

strychnine are usually given by mouth before the injection and if there is much pain there should be no hesitancy in giving morphine.

Dr. Charles G. Lucas, Louisville, Ky.—Crohn, in his book, says that "the chief function of the consulting physician is to make a rectal examination." It is a good thing to have our attention called to the rectum. If on our history sheets we had a certain place marked "rectal examination," we would save our patients a good deal of trouble.

Dr. D. R. Pickens, Nashville, Tenn.—I have used benacol in one case of pruritus. Pruritus is mostly due to infection in the tissues about the rectum or mucosa. It may be proctitis, colitis, sigmoiditis, papillitis or fissure or ulcer. If you cannot find any abnormality, these cases can be treated with quinine-urea injections which I use instead of benacol or hydrochloric acid. The patients are often highly nervous individuals and they are hard to relieve.

There is another type of infection involving the anus and crural region, namely, ringworm. No kind of injection will cure that; it has to be cured by the proctologist by some means, such as roentgen rays, and local applications, but do not try to cure it by injection.

I have cured indigestion by operating upon hemorrhoids and fissures, but I did not promise a cure. I believe rectal lesions produce nervous manifestations which cause indigestion. Relief of the symptoms which are producing the nervousness will relieve the indigestion. If a man has a fissure he is nervous and cannot digest his food properly. I never promise a man to cure indigestion by treating his rectum.

I refuse to operate upon a psychoneurotic, unless it is absolutely necessary. I usually have them come back to the office until they are tired and quit.

I have operated upon patients with cancer of the rectum who have had their hemorrhoids removed. I do not believe the medical profession overlooks all of them. I had a patient not long ago with an incurable carcinoma of the rectum, whose hemorrhoids had been removed. I think that occurs too often.

Dr. Curran Pope, Louisville, Ky.—I am a neurologist and an internist and I see the same kind of cases that Dr. Alvarez sees. In 1890 I heard the learned Charcot say, "Beware of the symptoms painted upon a neurotic background." I have never forgotten it. There are many psychopaths among women, men and also among physicians. I see them every day. Operation should not be recommended for them unless it is imperative. Their symptoms often by psychic control and education can be relieved so that the patients can adjust themselves to their environment.

I have recently, without a dose of medicine and without treatment, using nothing in the world but explanation and re-education, relieved an eczema that had resisted the onslaughts of medical men and dermatologists for fifteen years. This shows the tremendous power of a psychoneurosis.

Dr. James T. Wolf, Washington, D. C.—We must not forget that a number of these patients with rectal and digestive disturbances are women, whose symptoms may be caused by gynecological disorders.

Dr. F. M. Stites, Hopkinsville, Ky.—There is one thing that should not be overlooked, and that is food allergy. I do not believe any examination for a gastro-enterologic complaint is complete unless you make inquiry as to food allergy. There is hardly a symptom in the abdomen that cannot be simulated by some food allergy, from appendicitis to gall-bladder disease. I think we should always inquire whether it exists or not.

Dr. Hayes (closing).—I think it is very proper for Dr. Durham to emphasize the effect of rectal disease upon upper intestinal troubles. While it is not the whole cause, it is one of the leading causes, and, as has already been suggested, it is well to give a guarded prognosis in these causes. We find that in cases in which the rectal disease preceded the gastrointestinal symptoms, we are more likely to get a good after-result when the rectal condition is corrected.

Of course, whenever pruritus ani is mentioned, it precipitates discussion and, like indigestion and gall-bladder trouble and conditions of that kind, there are many remedies offered. Dr. Hanes said he had not used benacol very much. I am not so enthusiastic about its use as Yeomans, who claimed excellent results in the treatment of pruritus ani. With my patients, it has given only temporary relief. However, we have had cures in some cases of short duration.

Several years ago when Dr. Hanes first published his paper, I tried hydrochloric acid injections, but did not get as good results as he. I got some temporary relief, but did not think the results warranted its continued use. I have been greatly interested in Dr. Hanes' use of castor oil, of which I do not think he has yet made any published report. Not only from Dr. Hanes have I heard of this, but from other men. This summer at the meeting of the American Proctological Society in Detroit, Dr. Collins mentioned its use in bad cases of prolapse of the rectum. He cited one case in which the patient, a physician's wife, had had a prolapse for several years and was practically an invalid because of the condition. He sent her to the hospital and injected 10 or 12 ounces of castor oil around the rectum. This reduced the prolapse, and the case was entirely cured. I think anything that will entirely cure a bad prolapse is certainly worth trying, for certainly surgery in these cases is not always successful.

Dr. Durham (closing).—If I were a young proctologist, and should locate in a new place, and should my first three patients be suffering from pruritus ani, I would consider it to be a bad omen and I would leave the town. I know I could not make an enviable professional reputation treating pruritus ani. I mean pruritus ani as assocated with large anal folds covered with thick white friable skin.

I know nothing about benacol for postoperative relief of pain. I use a weak solution of quinine and urea hydrochloride injected under the skin and in the area of the incision, and obtain several days of anesthesia while the incision is healing.

I did not mean to create the impression that most gastrointestinal symptoms or disorders are caused by anorectal disease. However, some gastrointestinal diseases are made much worse by reflexes from the rectum upward. Most of the anorectal diseases are the result of conditions higher up in the gastrointestinal tract.

I have persuaded every internist and every diagnostician in the city of Columbia to use the proctoscope. I have accomplished this, and if I accomplish nothing more I am satisfied.

CHRONIC ENCEPHALITIS: STRAMONIUM TREATMENT*

By WALTER JOSEPH OTIS, M.D.,
New Orleans, La.

The epidemics of acute encephalitis lethargica have left in their wake sequelae which, for the most part, have been, and continue to be, the bete noir of the practitioner of medicine and of the neuro-psychiatrist in particular. This entity, likened to Proteus, has assumed forms, shapes and manifestations bizarre, grotesque and atypical in character. Symptoms, signs, onset, course of treatment have all been intelligently covered by neurologist and psychiatrist in monograph and text book, supplemented by didactic lectures in the class room and at the bed side, down to our present medical period, including the inexhaustible investigations of the Association of Research in Nervous and Mental Diseases, and the very illuminating volume of the Matheson Commission.

Epidemics have been recorded in ancient times, during the Hippocratic period, and down through the varied epochs of our civilization, with serious epidemics in 1712 in Japan, in Australia and Italy, and subsequent major epidemics in 1917 in Germany, Vienna and England. In March, 1919, this disease made its appearance in epidemic form in New York. Since that time it has continued to manifest its virulence within our communities, its greatest prevalence, however, being in Japan in 1924, with 6,949 cases.

For the condition of motor helplessness of these patients, atropine, scopolamin, hyoscyamin and their alkaloids have especially been used. The pharmacological actions of these we are fully apprised of, and also are we aware of their extensive use in the treatment of idiopathic paralysis agitans and following epidemics of encephalitis lethargica, Erb recording their value in 1906.

Babinski and Soukes recommended scopolamin for the sequelae of encephalitis lethargica, tremor, muscular rigidity, myalgia, ptyalism, and fatiguability. Hyslop, in 1907, reported its value in lessening the rigidity of post encephalitic syndromes. Within recent years a drug has been extensively used in the Parkinsonian syndrome, following acute encephalitis lethargica, which exerts a palliative effect upon these patients. It was known to the ancients as a poison, Laeanus mentions it in the early ages, and it was used in India for many centuries. This drug, the name of which is incorporated in my subject, is *Datura stramonium*, of the family solanaceae, an anodyne and antispasmodic employed for checking excessive salivation.

The first notice of stramonium is, in all probability, that of Fuchsius[1] (1524), who states that it was introduced into Germany from Italy. Gerard, who received some of the seeds from Lord Zouch, who sent them from Constantinople (about the Sixteenth Century), cultivated the plant in England. Baron Storck was the first to introduce the plant into medicine.

In 1779, Samuel Cooper[2] submitted a thesis on the properties and effects of *Datura stramonium* to the trustees of the University of Pennsylvania.

He mentions in part that the plant is frequently called thornapple, Jamestown or jimson weed, because a number of sailors were once violently diseased by ignorantly eating the boiled plant as potage in Jamestown, Virginia. In like manner it is called French chestnut in New Jersey. Cooper says that he has heard from various sources that the vapor emanating from the plant has a febrile action. He further relates that a soldier in the Revolutionary War was seized with a fever while lying under the plant, close by the River Wabash. The inhabitants of the place assert that previous to its introduction among them they knew not what remitting fever was. It is said that the weed in Jamestown was used as a salad by the soldiers. Those who had eaten plentifully thereof turned natural fools for several days; one would blow a feather in the air; another would dart straws at it with much fury; and another, stark naked, was sitting in a corner like a monkey, grinning and making mouths at them; a fourth would fondly kiss and paw his companions and sneer in their faces with a countenance more antic than any in the Dutch Droll. In this frantic condition they did play a thousand such simple tricks. This plant was commonly termed by the vulgar stinkweed, so says Ireland, the term originating from *Solanum fetidum*. Stramonium was known also to the Mohave and Pahute Indian tribes.

The particular treatment of the Parkinsonian state by stramonium was brought about by the monographs and experiences in the use of this drug by E. Juster,[3] Laignol, Levastine, Valance,[4] to be followed in America by Sophie Shapiro,[5] Jacobson and Epplen,[6] Carmichael and Greer,[7] J. Moren,[8] Holdemaker and Burns,[9] and J. H. Baird.[10]

The purpose of this essay today is to present results by the use of stramonium in the treatment of Parkinsonian state in a series of 20 cases post-encephalitic in character and type.

Of this series, 12 were white men, 8 of whom were married and had children. Eight were

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women, 5 of whom were white (2 of these pregnant) and one negro woman was included. The average age of onset in males was twenty-one and females twenty-three. These cases on first contact presented, for the most part, a lowered vital resistance with a lowering of the physical and mental morale, were undernourished, and in three cases underdeveloped.

All presented a typical Parkinsonian attitude, gait, station, facies, Cheshire smile, sialorrhea, with alteration in voice in varying degrees.

The interval between the onset of illness and the period of life when disability was most noticed varied from directly following convalescence to four years.

The anamnesis of the case was typically as follows: there was malaise, flushing of the features, headache, throbbing, ocular pain with some nausea and vomiting, fever which, to quote the patients, continued to a high degree, and in 6 marked delirium. A sleepy condition occurred with marked stuporous episodes varying from four to six to nine weeks.

On reacting to medical and nursing care, these patients would give a history as follows: "As I commenced to feel better, my body felt stiff. I did not feel like myself as before I took sick." Then followed an account of symptoms which led up to the continued treatment. They were the symptoms of a progressive motor helplessness with inability properly to handle and do for themselves as prior to the illness, with marked poverty of movement.

The atypical cases of the group I have briefly abstracted:

(1) There were tic-like manifestations of the head and neck, with grimaces in a white man of 34. Following investigation, this was found to be functional and ceased after several therapeutic interviews without additional medication.

(2) Hiccough was reported in a white woman of 29 of pronounced emotional disposition. This continued for three days at frequent intervals. Tincture of musk 30 minims was administered following a period of three days, when the distressing symptoms disappeared.

(3) A white female of 38 was pregnant. The usual prenatal hygiene and examinations were carried out during gestation. The patient proceeded to term and was delivered normally of a male child, without apparent accentuated after effects or lowering of her morale The child is living and well at two years of age.

(4) A pregnant white woman of 34 had the usual prenatal and maternal prophylaxis. A marked anxiety was externalized for the greater part of her gestative period. This was superimposed on a constitutional inferiority, plus an inadequate personality, all badly handled. A normal delivery followed, with a healthy baby.

(5) Three women, 22, 24 and 30 years old, previous to my contact were termed dementia precox, katatonic. These cases presented a marked rigidity, a negativism and severe oculogyric crises for periods varying from 1, 4 to 6 hours, one patient requiring nasal feeding on several occasions. Marked improvement followed treatment.

(6) A single white man of 34 had marked hysterical manifestations, plus a constitutional psychopathic personality, with accentuated emotional outbursts. These motivations varied from mild, moderate to severe during treatment. When last seen he was making a fair social adjustment.

A resume of the results of the medication was noted as follows:

Muscular hypertonia was relieved in all cases. Four regained almost normal postures and were able to attend to their various occupations; 4 were markedly improved to the extent of feeding themselves, when previously they had been almost bed-ridden and spoon fed; and 2 women showed slight improvement and were able to go about their homes unaided.

In disorders of movement all were benefited and the progress was on a parallel with muscular hypertonia.

Myalgia, a most distressing symptom accompanying this condition, was relieved to a gratifying degree in all cases in which it occurred.

Sialorrhea varied in degree in 14 cases. These showed gradual diminution to cessation of the condition and a more hopeful outlook and encouragement of the patient.

Four cases of marked oculogyric crises were relieved materially by medication.

Fifteen patients displayed a change of personality, reverting to their normal reactions, and were alert, interested, with facial expressions markedly changed, and a feeling of well being, confidence and hopefulness where at one time existed an abnormal emotional state, with irritability and crying, depressed coloring, and facial expressions of dejection and hopelessness. The remaining five had adjusted fairly well to surroundings.

Tremors had included all parts of the body, hands, tongue, eyes, lips and head, with marked facial tremors. They were markedly improved in 3 cases; improved in 14 cases; and slightly benefited in 3 cases.

POSOLOGY

The tincture of stramonium was used only 10 per cent, U.S.P., freshly prepared when possible. The average initial dose was 20 minims and average terminal 60 minims, three times a

day before meals, diluted with carbonated water when possible.

UNTOWARD EFFECTS

Xerostomia was present directly at the onset in all the series. It was relieved for the most part by sugar pellets, dragees, or gum chewing. It disappeared following the withdrawal of the drug for 24 to 36 hours.

The visual blurring usually experienced when nearly a maximum dose was reached was uncomfortable for a time now and then during treatment, but disappeared within 24 hours upon withdrawal of the drug.

Vertiginous symptoms were experienced in 6 cases when the peak of therapy was being reached.

Anorexia occurred in three cases only. These were given a tonic digestant as an aid for stimulating their appetites. This series exhibited no errors of the gastrointestinal tract as such.

Anxiety and apprehensiveness were noticed in four patients during medication. Two displayed language difficulties. When circumstances were explained to them, their emotional balance was restored.

RESUME

(1) Tincture of stramonium has a beneficial effect on the condition known as the Parkinsonian state or syndrome, following a history of chronic encephalitis.

(2) Medication to tolerance should be employed.

(3) The patient's morale appears to be materially helped by the administration of this drug.

(4) Untoward effects are not severe for the most part and are evanescent.

(5) Make every effort to remove all foci of infection, with attention to personal and mental hygiene, in conjunction with your treatment.

(6) Tincture of stramonium aids in replacing hopelessness by hopefulness.

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424 Chaille Building

DISCUSSION (*Abstract*)

Dr. R. Finley Gayle, Jr., Richmond, Va.—A number of years ago at this Section meeting in Atlanta, Dr. Gardner, of Louisville, read a paper on the sequelae of encephalitis under the use of hyoscine. I remember at that time that we were getting some objective improvement with hyoscine. It is, however, my opinion that stramonium is a better drug for this condition than is hyoscine. In approximately 75 per cent of the cases that I have been treating for more than a year with stramonium a rather marked improvement has been shown, particularly of the muscular rigidity, the tremors, and the headache. I have had, as has been Dr. Otis' experience, invalids who were bedfast or completely confined to chairs, who were improved very materially by the use of stramonium. Two patients I have in mind at this moment are now ambulatory and are feeding and dressing themselves. Others with fewer objective symptoms are getting along quite satisfactorily. Certain of the cases do not bear stramonium well, as Dr. Otis has said, and some others are showing no improvement.

We have seen a few patients with a post-Parkinsonian syndrome who showed some mental deterioration, others who showed an emotional flattening. We used the tincture of stramonium for a long period of time, but we have found that a preparation of the freshly prepared leaves gives better results. In all, I think that stramonium is a better drug in the treatment of the sequelae of encephalitis than hyoscine or other drugs which have been in general use.

Dr. Charles S. Holbrook, New Orleans, La.—Post-encephalitic Parkinson's syndrome has caused us a great deal of concern, for it is a most distressing condition, and any treatment that will lessen the severity of the symptoms should be received with enthusiasm. Hyoscine hydrobromate is beneficial, but there is no uncertainty in my mind that stramonium is even more efficacious. To a large extent, it relieves the tension and the tremor, and the patient experiences a feeling of well-being that he usually has not had for years. Stramonium does not seem to give as much relief in Parkinson's disease as it does in the post-encephalitic syndrome.

I have used freshly prepared tincture of stramonium, as it is most easily handled. There have been a few cases reported where druggists have substituted tincture of bella-donna for tincture of stramonium, with very serious results. Benefit is seldom observed unless large doses of stramonium are given. I find that 60 to 90 minims of the tincture should be given three to five or six times a day.

I have gotten far more satisfactory results from employing stramonium in post-encephalitic Parkinson's syndrome than from any other treatment.

Dr. Leslie B. Hohman, Baltimore, Md.—I hate to dissent from Dr. Otis' excellent presentation, but my experience after a number of years' work in this field has been radically different. Within the last two years I have worked with stramonium, but before that exclusively with hyoscine. In no case have I found that stramonium would relieve where hyoscine would not. My belief is that the failure to get satisfactory results with hyoscine has been due to the failure to use large enough doses. In severe cases I gave as much as 0.2 grain of hyoscine a day, that is, 0.04 grain five times a day. With this dosage I get much more relief of tremor and rigidity than I have ever been able to obtain with stramonium. To date I have not been able to substitute stramonium for hyoscine without causing the patient much discomfort and increase of symptoms. It may be that our fault lies in not prescribing large enough doses of stramonium.

Dr. Howard R. Masters, Richmond, Va.—I had been following the same method which Dr. Hohman has advocated in the use of hyoscine except that I have not been using as large doses. I finally began to use stramonium, which I like better. The large doses of hyoscine cause the patient at times much discomfort, dryness of mouth, drowsiness and sometimes nausea. These symptoms are absent in the use of stramonium and the effect seems to be much smoother. My choice of the stramonium preparation has been the 2.5 grain pill of stramonium leaves, giving the patient from two to six pills a day.

Dr. Carrol C. Turner, Memphis, Tenn.—I should like to ask Dr. Otis if he has had any experience with cystitis following the administration of this drug. Two of my patients who used it developed bladder symptoms. To make sure the symptoms did not develop from some other cause, I took them off stramonium and the symptoms cleared up. On the resumption of the drug the bladder symptoms came around again.

Dr. Otis (closing).—In reply to Dr. Hohman, on various occasions I have tried hyoscine in these cases only to find its demoralizing effect upon the individual even in that type of patient who is fundamentally intellectual. As to dosage, I had reference to minims only.

We are presenting this as a palliative and an ameloriant and not a cure-all. By this treatment we are able for the present to help and benefit the patients. Let us not forget the psychic factors in these cases. The fact that we substitute hopefulness for hopelessness, I think, is worth while.

SPONDYLOLISTHESIS, WITH SPECIAL REFERENCE TO THE INDUSTRIAL CASE*

By J. Edgar Stewart, M.D.,
St. Louis, Mo.

Spondylolisthesis, from the Greek, meaning slipping or gliding vertebra, is the term applied by Kilian in 1853 to the cases of forward displacement of the lumbar vertebrae on the sacrum. The term is descriptive and has not only remained in use, but is seen much more commonly in the literature in the past few years, due to the greater number of cases recognized clinically and to their importance as disabling factors in industrial accidents. The credit for the more frequent recognition is due entirely, I believe, to improvement in radiological technic, making lateral views of the lumbosacral region obtainable in which the outline of the lumbar and sacral bodies are plainly visible even in large and obese subjects. The fact that 5 of the cases reported here were diagnosed at one railroad hospital during the past two years and that about this time the routine was established of taking lateral films of the lumbosacral region of all patients with symptoms referable to the low back, make the above statement fairly obvious.

PATHOLOGICAL ANATOMY

The pathology of spondylolisthesis and the mechanism of its production were studied before roentgenology by the examination of dissected specimens. These and subsequent anatomical and radiological studies bring forth certain important points associated with the condition that are fairly well agreed upon. (1) A separation of the neural arch is associated with its occurrence. (2) Disarticulation or unlocking of the articular facets does not occur. (3) There is an antero-posterior elongation of the fifth lumbar vertebra.

The vertebra develops from three centers of ossification: two for the posterior portion and one for the body. The vertebral arch should become complete during the first year, but does not become joined to the anterior portion till some time later. When complete fusion of the anterior and posterior portions fails to occur, it becomes an important factor in predisposing to

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spondylolisthesis. Chiari, in 1892, demonstrated by careful examination of anatomical specimens of spondylolisthetic spines that a symmetrical non-union of the laminae occurred in such cases, but did not make it clear that they were developmental defects. A failure of ossification is not infrequently found, however, in dissections of vertebrae without existing deformity. It is impossible to determine such a condition clinically, or it might be an important medico-legal point in industrial cases. Mueller believes that all cases of spondylolisthesis have this congenital defect as their basis. This claim would be hard to substantiate, as the thrust of the superimposed weight of the body of the fifth lumbar vertebrae in some of the cases presented here would seem sufficient to fracture the pedicle of a perfectly ossified vertebra. It is important to note that the displacement is not of the fifth or fourth lumbar from the remaining vertebrae, but of the entire vertebral column on the sacrum or fifth lumbar as the case may be. Turner and Tchirkin maintain that the spinous process of the displaced vertebra is not carried forward with its anterior segment and that the hollow above the sacrum is caused by the tilting upward of the affected spinous process. I am inclined to agree with Kleinberg that the spinous process of the slipping vertebra is carried forward by its attachment to the spinous process of the vertebra above. There is no question that the spinous process of the slipped vertebra is usually tilted upward as seen in antero-posterior radiograms in most cases.

INCIDENCE

Early writers on this subject believed that spondylolisthesis was a very rare condition occurring almost exclusively in women and taking place usually during parturition. It is now almost certain that it occurs more frequently in men, that severe trauma is an important factor in its production, and that it is a fairly frequent occurrence. Sir Arbuthnot Lane believes that excessive weight bearing or heavy labor over a long period of time brings about the displacement. He considers it a normal occurrence in heavy laborers such as coal heavers. My examination of a large number of lateral views of lumbosacral spines of men doing heavy labor does not bear out this view. Such often repeated trauma may be the cause in some instances, however (one of Wilson's cases) and more frequent lateral radiograms with the patient standing might alter our opinion.

CLINICAL PICTURE

The diagnosis in the cases with exaggerated displacement, or those of long standing, can be made from the clinical picture: the shortened lumbar segment with lordosis; tender prominence of the first sacral spine with a hollow space above it; sometimes a waddling gait somewhat like that of bilateral displacement of the hips but less exaggerated. These are seldom present in the acute cases with moderate displacement, and it is just such cases that the industrial surgeon must detect at the earliest possible time. Symptoms complained of are usually those of any acute back strain; pain in the low back, probably extending down the backs or fronts of the thighs; inability to be long on the feet or to lift. In the acute cases in this series, marked tenderness in the lumbosacral region was a constant sign, as was restriction of motion, pain on motion of the spine and muscle spasm. In none of the cases was there evidence of damage to the cauda equina or nerve roots.

The conclusive evidence is in the radiological examination and without radiograms the existence of the displacement can only be guessed at. Any patient complaining of low back disability or one who has sustained an injury to the back should have the benefit of a radiological examination, and it is my opinion that radiograms in two planes are just as important in this region as they are about the knee, ankle, elbow or wrist. The importance of a radiological examination at a later date, in cases where the examination following the injury was negative and where symptoms persist, is strikingly brought out in one of the cases reported here. Radiograms the day following the accident were negative and symptoms were not significant except for acute lumbosacral strain. After the patient had been on his feet for some time symptoms increased and radiograms made about two months after injury showed the fifth lumbar body slipped forward about one-half inch on the sacrum.

TREATMENT

The treatment of these cases, where the diagnosis is made soon after injury, is that of any other acute back injury, continuous recumbency on a Bradford frame or hard bed. The writer has been discouraged from trying manipulative reduction by the reports of competent men who have tried it unsuccessfully, together with the fact that only one case has been seen soon enough after injury to make attempted reduction practical. The condition is usually an extremely

disabling one, and particularly in industrial work the writer has unreservedly advised operative fusion of that portion of the spine. Where operation is refused, a non-elastic corset is applied, and it is recommended that the man should not be returned to work that requires lifting, running, jumping or any strain on the back.

When undertaking operative fusion for this condition, it is well to recall again that the entire spinal column is displaced forward and that the ligaments normally relied on to hold the structures in position have been stretched or torn. An unusually firm and solid bridge of bone, extending from the sacrum to the fourth, or even the third and fourth lumbar vertebrae, is essential. It can readily be seen that a bridging from the sacrum to the posterior portion of the fifth lumbar alone would be entirely ineffectual. I have used the Hibbs procedure and added bone grafts laid along the laminae on either side. The crest of the ilium has been found a convenient place to get grafts for this purpose.

CASE REPORTS

Case 1.—R. S. A., a woman, age twenty years, was seen at the orthopedic dispensary, Washington University, complaining of a weak back and a lump on the back. She said her back had been weak and painful since her only child was born three years before she applied for treatment. She had tried to work as a salesgirl, but found it impossible to stand on her feet for any length of time on account of the pain in her back. Stooping was difficult and she had no strength for lifting.

On standing, the trunk was shortened, the waist relatively high. There was lordosis with a sharp angulation in the low lumbar region. She walked with a waddling gait somewhat like that of a bilateral dislocation of the hips. There was a marked prominence of what appeared to be the first sacral spinous process with a somewhat tender hollow space just above it. Motion of the spine was restricted in all directions. Antero-posterior radiograms showed a tilting upward of the spinous processes of the fourth and fifth lumbar vertebrae. Lateral radiograms showed the body of the fifth lumbar displaced completely forward and downward from the body of the first sacral. A bilateral separation of the bases of the laminae had apparently taken place. Operative fusion of the lumbosacral spine was advised. She was admitted to Barnes Hospital and an anterior and posterior shell was made for her to lie in. July 13, 1926, a Hibbs fusion of the sacrum and fifth and fourth lumbar vertebrae was performed. She was recumbent in the plaster shell eight weeks. A walking jacket was applied and she was allowed up for increasing periods. She was last seen October 25, 1926. She walked into the clinic and stated that she no longer had the weak feeling in her back.

Case 2.—L. D., age sixty-seven, a railroad section foreman, was admitted to the Missouri Pacific Hospital October 18, 1930, complaining of pain in the back and down the back of the right leg and shortness of breath. He said that twenty-seven years previously he was struck in the back by a moving car. He was taken to a hospital in Kansas City, where a plaster jacket was applied, and returned to work as a section worker several weeks later. He had had recurrent attacks of back pain since then, but was always able to relieve it by rubbing with liniment until the present attack, which started three months before.

The patient stood with a right lumbar, left dorsal deviation. He was tender in the lumbosacral region and there was a depression in the lumbosacral region with prominence of the first sacral spinous process. Motions of the spine were greatly restricted in all directions. Antero-posterior radiograms showed exostosis on the bodies of the lumbar vertebrae. There was a deformity of the second lumbar vertebra with angulation of the lumbar spine at this point. Lateral view showed a line extending across the laminae of the fifth lumbar and a displacement forward of the body of the fifth lumbar on the sacrum. A non-elastic corset was ordered and it was recommended that he be taken off work that required any stooping or lifting.

Case 3.—P. F. G., a man of sixty-four, a bridge builder, was admitted to the Missouri Pacific Hospital for attacks of dyspnea, swelling of the extremities and pain in the region of the heart. Routine history brought out a complaint of a periodically painful attack since a fall twenty-one years before admission. The back condition in no way incapacitated him for his work as a bridge builder (heavy labor).

Examination showed a tender point in the lumbosacral region slightly to the left of the midline. Spine motions were restricted in all directions, but not by pain or muscle spasm. Lateral x-ray showed displacement forward of the fourth lumbar on the fifth. As this patient was handicapped by a cardiac condition, no treatment was recommended for the spondylolisthesis.

Case 4.—Martha S., a woman of fifty-one, a housewife, was seen November 21, 1927, at Barnes Hospital. She had been admitted to the Hospital for severe pain in the back and inability to be on her feet for any length of time. She was sometimes conscious of a slipping sensation in the low back, followed by severe pain and disability. There was no history of injury, but she said she had had pain in her back since she was twelve years old. She had had three children.

The spine was lordotic. She had a large, pendulous abdomen and heavy torso, and a tender prominence of the first sacral spinous process. Motions were restricted in all directions and guarded by muscle spasm. Antero-posterior x-rays of the lumbosacral spine showed a tilting upward of the fifth lumbar spinous process. Lateral views showed a slipping forward of the body of the fifth lumbar vertebra on the sacrum about one-half inch.

Spinal fusion operation was advised, but she could not be away from her family to have it performed, so a non-elastic corset was supplied. Correspondence six months later stated that she had been much benefited at first, but that recently she had had the slipping sensation again and was able to be on her feet only for short periods.

Case 5.—George P., a man of thirty-eight, a switchman, was admitted to the Missouri Pacific Hospital December 16, 1929. He was thrown from a flat car November 30, 1929, striking his back against a draw bar, and had had pain in the low back and inability to stand for long at a time since.

Examination showed a tender depression just above a prominent first sacral spine. The spine was held rigid by muscle spasm and attempted motions were painful. Lateral radiograms showed a displacement forward of the lumbar spine on the sacrum.

The patient was put on a hard bed and heat applied to the back for four weeks. A fusion operation was advised, but refused. A canvas pelvic belt was fitted and it was recommended that he be taken off work as a switchman. Recent attempts to get in touch with this patient have not been successful.

Case 6.—Perry H., a man of forty-eight, a locomotive engineer, was admitted to the Missouri Pacific Hospital October 30, 1929. October 29, 1929, the day before admission, he jumped from his engine cab when about to collide with another train. He did not know how he struck. He was able to stand and walk, but his low back was very painful.

Examination showed tenderness in the lumbosacral region. No prominences nor depressions were felt. Motions of the spine all were restricted by muscle spasm. Antero-posterior and lateral radiograms showed no injury nor displacement of the vertebrae.

He was kept recumbent on a hard bed for ten days, when symptoms seemed to have subsided, and he was allowed to return to his home. He returned in April, 1930, complaining that his back became painful on standing or sitting and that riding in his cab was extremely painful. He was unable to lift or stoop. Radiograms taken at this time showed a displacement of the lumbar vertebrae on the sacrum about one-half inch. A non-elastic belt was applied and he was advised to keep off his feet and avoid stooping and lifting. A communication on October 3, 1930, states that he is no better than when last seen, and has been unable to work.

Case 7.—George R., a man of forty, a freight house laborer, was admitted to the Missouri Pacific Hospital October 5, 1929. He said he was struck in the back by a loaded hand truck and jammed against a door. He had been unable to work since on account of pain in the low back when he stooped or lifted.

Examination October 29, 1929, showed tenderness in the lumbosacral region and motions of the spine restricted by muscle spasm. No prominences nor depressions were felt. Lateral radiograms showed displacement forward of the lumbar vertebrae on the sacrum. On account of some service ruling, this man was discharged from the hospital and efforts to get in touch with him have not been successful.

Case 8.—Chas. K., a man of forty, a car cleaner, was admitted to the Missouri Pacific Hospital on May 17, 1930, shortly after having been struck by a locomotive and knocked down.

Examination the following day showed tenderness of the entire spine from occiput to sacrum, painful on attempted motions of the spine. Radiograms showed a fracture of the spinous process of the fifth cervical vertebra and a forward displacement of the lumbar vertebrae on the sacrum. He was kept on a hard bed for eight weeks, receiving physical therapy. At the end of eight weeks a non-elastic corset was fitted and he was allowed up for gradually increasing periods.

Examination October 30, 1930, showed tenderness in the lumbosacral region and restriction of motions of the cervical spine. The patient seemed depressed mentally and complained of headaches and ringing in the ears.

Case 9.—Forest R., a man of thirty-two, a brakeman, was admitted to the Missouri Pacific Hospital September 30, 1927. He gave a history of falling between the freight cars of a moving train and of having been knocked to the side of the track. He was not unconscious.

He was very tender over the entire lumbosacral region. All movements of the spine were very painful and guarded by muscle spasm. There was a prominence of the spine in the lumbosacral region. Antero-posterior radiograms showed a fracture of the transverse processes of the fifth lumbar vertebra on each side. Lateral views showed a posterior displacement of the lumbar spine on the sacrum (reverse spondylolisthesis). He was placed on a hard bed and kept recumbent for four weeks. During this time he had radiant heat on the low back one-half hour twice daily. At this time a fusion of the spine was suggested as a possibility. At the end of six weeks he was allowed to be up for increasing periods, wearing a non-elastic corset. Recent efforts to communicate with him have been unsuccessful.

Case 10.—Man, age forty-two, a welder, was admitted to the out-patient department of the Missouri Pacific Hospital complaining of pain in the back and difficulty in stooping since a fall on the stairs at home three weeks before admission. He said that he had had a severe accident to his spine when thrown from a sled when a boy 14 years old. He was in bed for a time, but had had no subsequent trouble in his back until the present time. He said he had been nicknamed "Kangaroo" on account of his waddling gait.

His trunk was shortened and spine seemed to sit low between the ilia. There was a definite prominence of the first sacral spine with a hollow above it. There was no tenderness at this point. Motions of the lumbar spine were considerably restricted. Lateral radiograms showed a displacement forward about one inch of the lumbar vertebrae on the sacrum. This man's work was done sitting or lying down and did not require lifting or jumping. A fusion of the spine should be considered if he were going to do heavy work.

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DISCUSSION (*Abstract*)

Dr. W. B. Owen, Louisville, Ky.—Many of these cases are not diagnosed because it is only possible to make the diagnosis absolutely, unquestionably, if a good lateral x-ray picture is made. The anterior-posterior alone, which we see used many times, is not sufficient. It will not show the conditions which exist.

I used to have an idea that it would be possible to make a partial reduction, but have gotten that out of my mind. Traction, counter traction, flexion, exten-

sion and other manipulations have never been of value in my hands. The only thing that is helpful is fusion. Very few cases are cured so that they are capable of returning to hard labor and carrying on as they did before they had the injury. Many times the condition has existed for many years before some trauma or sudden fall produces disability. It is a potential liability, like a cervical rib or sacralized fifth lumbar. As Dr. Stewart has outlined, the accepted treatment at this time is fusion of the lumbosacral joint, building up a large foundation from the sacrum, including the fifth lumbar, the third lumbar at least, and the second, and sometimes the first. Even then these cases should have a spinal brace for at least a year.

It is very seldom that a spinal brace or splint is sufficient to relieve severe cases of spondylolisthesis.

Dr. Custis Lee Hall, Washington, D. C.—At the Veterans' Hospital we x-ray spines almost routinely, and possibly in the last few years some two or three thousand spines have been examined. At times in cases of this type we get no history whatever of any previous injury, and some patients have no symptoms of disability in their spine, no pain, and no stiffness. One patient had a second and fourth lumbar vertebra which had evidently slipped forward.

Dr. I. M. Flinn, Jr., Wilmington, Del.—As a house officer in New York I saw several of these cases. One of them was in a child of eight. She had a slipping forward of one-half the distance of the body, with pain. I wondered whether the condition of the woman Dr. Stewart showed was not probably a congenital affair from the start, and more or less of the same type as this little girl.

Dr. Ferguson, Roentgenologist at the Hospital, has been studying these conditions. He feels, I understand, that the posterior dislocation has little or nothing to do with trauma. He thinks probably it is a congenital lesion rather than a traumatic one.

While there I saw also two cases that had to be re-fused and one was fused a third time. There was probably not enough bone at hand in those cases and considerably larger reinforcement of the spine fusion itself was indicated.

Dr. Stewart (closing).—The question of the congenital origin of these things, whether there is a congenital failure of fusion or a weakness at least of the portion of the vertebrae where it joins the body to the posterior portion, has been under discussion since spondylolisthesis was first recognized.

I think congenital weakness undoubtedly is the cause of a certain number of cases. I do not know any way to determine how many of them. The fact that a number of these cases were big, heavy men who had severe trauma, such as a fall from a height, in all probability striking on the buttock or the back, makes the condition, for the purpose at least of industrial boards, traumatic in origin.

Even if the patients have a congenital defect, if they have had a good, useful spine, and have been able to do hard work during their lives previously, then I think the condition must be considered as traumatic.

BRUCELLA ABORTUS INFECTION IN MAN: CLINICAL MANIFESTATIONS AND DIAGNOSIS*

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Within the past six years an increasing number of scientific studies and observations of undulant fever have been made in many sections of the United States, and there has been a renewed interest in a number of European countries.

This infection in man was first described in our country by Colonel Craig, of the United States Army, in 1903. There were no further studies until some ten years later, when Larson and Sedgwick carried out their experiments and studies of contagious abortion in cattle, and in their conclusions suggested that this organism might be pathogenic for man. In 1924, Evans made serological studies on 500 human serums from a variety of febrile diseases, and again the cow was suspected as a possible source of this infection in man. In 1924, Keefer reported the first case in America caused by the *Brucella melitensis, variety abortus*. There has been a marked increase each year in the reported cases since 1926, and in 1929 one of our Western states reported 125 cases caused by *Brucella melitensis, variety abortus*.

Undulant fever has become an important clinical entity, but it has not received the recognition by the profession that it deserves. It has been said that undulant fever is a disease of the future. However, it is obvious that the disease is not a new one, but one newly recognized. Standard medical books, monographs and current literature describe this disease under a multitude of names. From its geographic distribution, the disease has received the names of Malta fever, maltes, Mediterranean fever, Gibraltar or rock fever. Some authors describe the disease as Neapolitan or Cyprus fever. In Texas and New Mexico it is called Mediterranean phthisis from the bronchitis, anemia, loss of weight and night sweats which are suggestive of phthisis.

Captain M. L. Hughes, of the British Army Medical Staff, in 1897, proposed the name of

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undulant fever for this disease. This name was recommended by the International Congress of Medicine held in London in 1913. In view of the wide dissemination of the disease, the term has been generally adopted for the condition in man.

The etiology of Malta fever remained unknown for many years. In 1887, Colonel Bruce isolated an organism from the spleen of a soldier who had died of Malta fever. The next year he was able to grow this organism and to establish it as the etiological factor in Malta fever. The name *Micrococcus melitensis* was given the newly discovered organism. Bruce's work was soon confirmed by others. During the work of the British Mediterranean Fever Commission, the goat was discovered to be the source of this infection in man.

In 1896, Bang, of Denmark, isolated the organism that caused infectious abortion in cattle and soon after this organism received the name of *Bacillus abortus*. The organism of Bruce and the organism of Bang did not receive any further study until some twenty years later. To Miss Alice Evans, of the United States Public Health Service, belongs the credit of demonstrating that man is often infected with the organism that causes contagious abortion in cattle and hogs. In 1918, Miss Evans carried out experiments showing that the organism of contagious abortion (*Bacillus abortus*) in cattle and other domestic animals is indistinguishable morphologically, culturally, biochemically and by the ordinary agglutination tests from the organism that causes Malta or goat fever, the *Micrococcus melitensis* of Bruce. This discovery of Miss Evans has been repeatedly confirmed by others and with this the newer knowledge of undulant fever begins.

A reclassification and a renaming of the organisms was brought forward by Meyers in 1920. The name of brucella was proposed in honor of Colonel Bruce. This suggestion was readily approved and the name *Brucella melitensis* has been universally adopted. However, previous distinctions are maintained by the recognition of the different varieties: *Micrococcus melitensis* became *Brucella melitensis, variety melitensis* or *variety A*, and the *Bacillus abortus* became *Brucella melitensis, variety abortus*. The latter variety has been divided into the bovine and porcine types.

Morphologically, the *Brucella melitensis* is a small bacilliform or coccoid organism, non-encapsulated, non-motile, gram-negative and not forming spores. Culturally, its growth is best at 37° C. The organism is difficult to grow and growth takes place very slowly in liquid media, rarely earlier than the fourth day and often not until the fourteenth day or later. The optimum hydrogen ion concentration is pH 6.8. Huddleston reports better success with a liver infusion broth and agar. The porcine type grows best in an atmosphere of 5 to 10 per cent carbon dioxide. On dextrose agar slants, the colonies appear small, "dew-drop like," and when older, they have a light cream color.

The natural habitat of the *Brucella melitensis, variety melitensis*, is in the goat. The *Brucella melitensis, variety abortus*, may be found in a number of domestic animals and, from reports, undoubtedly all of our states and many other countries have cattle, hogs and other domestic animals that are infected with the *Brucella melitensis, variety abortus*. The porcine type of the abortus variety was isolated in 1914 and recently this type has been given careful study. The controlled experiments of Mitchell have shown a number of interesting facts:

"First, the *Brucella abortus* is not found in the uterus except during pregnancy and for the first few weeks following delivery. Second, the infection usually takes place by the alimentary tract and artificial infections may take place through the udder, the mucous membrane of the eye, subcutaneous and intravenous routes, but, so far, the attempts by the vaginal route have been negative. Third, infected animals may abort once or twice, but the infection does not necessarily clear up and may persist for the life of the animal. Fourth, abortion does not take place in each infected animal; an animal infected or harboring the organisms in the mammary gland or the uterus during pregnancy may never abort. Fifth, pregnancy is not necessary for infection. Sixth, the mammary gland and adjacent lymph nodes are the only tissues which have been shown to harbor the organisms in the absence of pregnancy, but surprisingly few histological changes take place in these tissues."

The clinical manifestations of this disease are frequently confused with a number of rather common maladies: typhoid, malaria, tuberculosis and influenza, and less commonly with rheumatism and bacterial endocarditis. Often the clinical picture is so indefinite that the clinician must look to the laboratory for the diagnosis. The incubation period has not been definitely established because we do not know when the infection occurs. Some authors state that it varies from six to fourteen days. The animal incubation period varies from eight to twelve days.

The early symptoms are vague and indefinite and the first symptoms are usually those of a general infection. The initial complaints are weakness, fever, headache, chills and sweating. Later, the patient becomes aware of an after-

noon temperature associated with backache, headache, muscle and joint pains, and a general malaise. Perspiration may be noticeable and may be of a peculiar sweetish odor. The majority of the cases that we have studied did show the above symptoms. However, they appeared comfortable, mentally alert and often said that they felt fine, although there are cases that are quite ill and remain so for weeks or months. The tongue is coated and a congestion of the throat is not uncommon. Bronchitis and a cough are frequent symptoms. The heart action is in keeping with the condition and there is no complication. The blood pressure is normal. The spleen is palpable in the majority of cases and the liver is congested. The temperature record is of particular value in undulant fever. A definite type of fever curve is recognized, but the undulant type of fever curve is not constant, and often the patient will run a low temperature with an afternoon rise. The temperature may be intermittent or remittent in type, ranging from 98° in the morning to 101° in the afternoon. The blood shows a moderate anemia with a slight to a moderate leucopenia. There is a decrease in the polymorphonuclears with an increase in the large and small lymphocytes. The hemoglobin is decreased. The urine shows very little change. There is usually a loss of appetite and some intestinal disturbance.

The *Brucella abortus* infection in cattle shows two distinct features and they are: abortion in cows and seminal vesiculitis in bulls. Simpson and Frazier report 5 cases in women who have miscarried repeatedly. None presented any evidence of syphilis and all gave histories of previous infections with *Brucella abortus*. The agglutination titer ranged from 1-80 to 1-320 and all had been consumers of raw cow's milk. Also, they reported 3 male patients who were treated by a genito-urinary specialist. The 3 patients had conditions which simulated gonorrhea, but repeated smear examinations from the discharge and prostatic secretions did not reveal positive evidence of gonorrhea. The agglutination tests for these three patients were positive for *Brucella abortus* infection.

CASE REPORTS

Case 1.—E. H. D., a man of 32, well developed and well nourished, an insurance agent, had resided in the city all his life. March 6, 1929, I was requested by his physician to see him and make a number of laboratory tests. The patient had been sick for twelve days. His symptoms were weakness and a tired feeling. His temperature was 102 to 104° F. He had a chilly feeling, but no definite chills. He had headache and pains in the back and chest. He was nauseated at times. There was no history of the patient's coming in contact with any domestic animals. On the afternoon of March 6 he was admitted to the hospital with an undetermined diagnosis.

Laboratory Examinations.—The blood count was: erythrocytes, 4,120,000; leucocytes, 7,000; hemoglobin, 82 per cent; differential, polymorphonuclears, 47.5 per cent; lymphocytes, 49.5 per cent with an increase in the large lymphocytes. Malaria examination was negative. The Widal, paratyphoid A and B, was negative. The undulant fever test was very strongly positive. Blood culture was negative. This patient remained in the hospital for ten days. All symptoms cleared up except the fever. Three months from the time the diagnosis was made he was well clinically.

Case 2.—S. J. S., a man of 48, well-developed and fairly well nourished, was a grocer. He was a resident of the country. He had been ill for more than two months and various diagnoses had been made. His symptoms were aching all over, weakness, chilly feeling, afternoon temperature, cough and heavy night sweats.

Laboratory Examinations.—There were 4,850,000 erythrocytes, 4,800 leucocytes, 93 per cent hemoglobin, 42 per cent polymorphonuclears, 57 per cent lymphocytes. Malaria examination was negative; Widal, paratyphoid A and B, negative; undulant fever test, positive; blood culture, positive. This patient was ill about eight months. Weakness and an afternoon temperature were the principal symptoms. He was a heavy consumer of raw cow's milk, but gave no history of contact with goats. He had fully recovered and was free of fever ten months from the onset. In the last report that I had this patient was enjoying good health and was at work.

An accurate diagnosis of undulant fever may be made by growing the specific organism from the blood or the urine or by demonstration of the specific antibody in the patient's blood. On account of the difficulty of growing the organism, failure is of little or no significance. However, a positive blood culture of the specific organism is diagnostic of undulant fever.

Blood for cultures should be taken at the height of the paroxysmal temperature wave and, preferably, in the afternoon. Ten c. c. or more of blood should be planted in one or more flasks of fresh beef liver infusion, hydrogen ion concentration pH 6.8. Better results are obtained if two or more different culture media are used and varying amounts of blood are added to the media.

Blood received in Keidel tubes may be cultured by removing the clot aseptically and planting it in 50 c. c. of fresh infusion broth. All cultures should be incubated from four to fourteen days, and negative cultures longer. After the fourth or fifth day, frequent examination of the cultures should be made by transplanting some of the media from the bottom of the flask to a dextrose agar slant. For the first two hours in the incubator the tubes should be so placed that the broth covers the slope. If the *Brucella melitensis* organisms are present in the culture, they will appear on

the slants in small "dew-drop"-like colonies after two or three days' incubation.

Urine for cultures should be collected aseptically and a number of dextrose agar slants inoculated, using 2 to 3 c. c. to each tube. For the first two hours in the incubator, the tubes should be so placed that the urine covers the slopes. Cultures should be made daily over a considerable number of days. If a growth appears resembling the *Brucella melitensis*, transplants should be made for further identification. When the blood is collected for cultures, enough should be collected for the agglutination tests, as this is one of the most valuable tests in the diagnosis of undulant fever. For many years serological methods have been used for diagnosing this infection, and indeed only by serological methods may the majority of cases be diagnosed. Antigens used in the agglutination tests must be tested with positive serum to make sure the antigen will be agglutinated. We find antigens made from the abortus variety to be more satisfactory than antigens made from any other varieties. There are two methods used in determining the agglutination reaction. (1) The slide or spot method as described by Huddleston is used by a number of laboratories and it consists of adding a small quantity of serum to a drop of concentrated antigen and noting the change, if any. Serum showing any degree of agglutination should be checked by the tube method. (2) In the tube method, a number of small tubes are used and various dilutions of the sera are made with normal saline. A good plan is to begin with a low dilution and gradually go to a higher dilution. One-half c. c. of the several diluted serums are placed in tubes and 0.5 c. c. of the antigen is added to each tube. The tubes are placed in the water bath at 37° C. for three hours, then in the refrigerator for 18 to 20 hours. Then the tests are ready to read. Agglutination in dilutions less than 1-40 are regarded as negative; in dilution 1-40 doubtful; 1-80 are read as weakly positive. Dilutions of 160 and 320 are read as positive reactions. Dilutions of 600 and 1,200 showing agglutination are read as strongly positive and those above this as very strongly positive.

Negative findings do not prove the absence of the infection, as there are authentic cases where the organisms were cultivated from the blood and the agglutination tests were negative or only slightly positive. Evans and Carpenter each have reported cases of this nature. Then there is a non-specific reaction which has been found in normal sera and more frequently in acute febrile diseases. Tularemia is a disease that will frequently give a positive reaction. Serums from suspected cases of tularemia should be tested with the *Bacterium tularense* antigen.

The intradermal reaction for the diagnosing of undulant fever is certainly a valuable aid, but we have not given it a thorough trial.

The pathologic changes in man as observed are far from being complete. There have been few autopsies and until further studies are made the pathology of this disease will be obscure. Most cases show an enlarged spleen and liver. There may be a basal congestion and a bronchitis of both lungs. Occasionally there is an involvement of the intestines with an enlargement of the lymph nodes. Orchitis and a mucopurulent discharge have been observed. The blood shows a moderate to a severe anemia. Hemoglobin is reduced. There is a slight leucopenia with an increase in the lymphocytes. The kidneys are negative and there is usually some loss in weight.

Pathological change in domestic animals has been studied more thoroughly than the changes in man. Nearly all animals may be affected, including the cow, sheep, goat, hog, horse, and others. In the cow there are acute, subacute or chronic inflammatory changes of the chorion and the endometrium. The udder shows a diffuse cellular infiltration, which usually persists through the life of the animal. The supramammary lymph nodes are enlarged and show a diffuse cellular infiltration. After abortion, there is a muco-purulent discharge and the bacteria are found in the milk and urine in small numbers. The milk and urine of the goat show the organisms in large numbers.

There are a number of common maladies that simulate undulant fever. The laboratory test that is used most extensively is the agglutination reaction. However, this cannot always be relied upon, as the agglutination can be obtained in low dilutions with normal serum and some typical cases of undulant fever never show agglutination in dilution above 1 to 5.

It should be remembered that the only sure diagnosis is a positive blood culture; that the infection is not uncommon; that the causative agent is identical with the organism causing contagious abortion in cattle; and that raw milk from cows infected with contagious abortion is dangerous for man.

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DISCUSSION (*Abstract*)

Dr. Foster M. Johns, New Orleans, La.—The infectious nature of cattle abortion has been known since 1567, and it then required 344 years of medical science to recognize that occasionally a human being may become infected. Dr. Ayres is quite correct in characterizing undulant fever not as a new disease, but merely as one newly recognized.

Modern medicine is slowly beginning to evaluate a number of diseases commonly prevalent in domestic or field animals which apparently only incidentally infect man. In a recent book by T. G. Hull on "Diseases Transmitted from Animals to Man," I was astounded to note that out of 16,000 examinations of milk, 8.6 per cent contained virulent bovine tubercle bacilli; and that 30 per cent of tuberculosis in persons up to the age of 16 years was due to the ingestion of this infected milk. Now that we can all add a few cases of *Brucella abortus* infection from infected milk and butter, we had better form a closer liaison with the veterinarians and not forget such other diseases as actinomycosis, milk sickness, foot and mouth disease, anthrax, distemper, ringworm, diphtheria, rabies and the host of other such diseases that altogether amount to an overwhelming total.

Economically speaking contagious abortion of cattle has been determined to reach the enormous total of 80,000,000 abortions in cattle annually, and 20 to 40 per cent of cattle are, and have been for an unknown period of time, infected in all parts of the country. In man it is quite doubtful that even if proper tests were made in all probable cases a very great number of infections would be disclosed. On the other hand, it certainly is gratifying to be able now properly to diagnose the few cases that have puzzled many previous consultants with their long continued fevers and sweats, and to whom quinine or hexylresorcinol has been given until the patient himself has rebelled. I am reminded even of a physician who after five months of agonizing massage treatment of a supposed prostatitis, finally consulted an internist to find that he had undulant fever with seminal vesiculitis incidental to the infection with *Brucella melitensis var. abortus*.

Dr. Ayres' paper is quite timely, the case histories are interesting and the review is fairly comprehensive. I want to disagree with him in two particulars only:

First, while the natural habitat of *Brucella melitensis* is the goat, we certainly have undoubted implantation of this strain in our cattle infections. Some of our cases give a very high titre agglutination reaction with this strain, and produce only a slight cross agglutination with the *Brucella abortus* strain. Clinical laboratories should routinely use both strains. There is considerable evidence that the type of human disease with the two strains may vary to the extent that the variety of the strain infecting the patient may occasionally be foretold.

Second, I believe that complete agglutination and sedimentation of the bacteria in question in 10 to 12 hours in much lower dilutions than he stipulates is coming into more general acceptance. When viewed in the light of other clinical and laboratory data a positive agglutination at even a 1-20 dilution should warrant at least a presumptive diagnosis.

Stool cultures and cultures of prostatic or vesicle secretion according to the method of Amos and Poston of the Johns Hopkins should be included in the diagnostic armamentarium.

Dr. Alfred Blumberg, Oteen, N. C.—The *Bacillus abortus* of Bang had been considered to belong among the microorganisms not pathogenic to man. In addition to the many communications found in the more recent literature, we have the assurance to the contrary of Dr. W. E. Cotton, Superintendent of the Experimental Station, Bureau of Animal Industry, Bethesda, Maryland, who informed me of several infections which occurred among meat inspectors who handled diseased animals, and of one laboratory infection which occurred in a technical helper. One may assume that the *Micrococcus pseudo-melitensis* of Sergent and Zammit and the *Micrococcus para-melitensis* of Negree and Raynaud were identical with *Brucella abortus* of Bang. Dr. Cotton compares the Brucella type of organism with the tubercle bacillus, stating that just as one speaks of the human, bovine and avian types of tuberculosis, one may speak of undulant fever of the Bruce, Bang and Traum type. I should propose the use of the term "Brucelliasis" to designate the clinical form caused by either of these organisms. For the purpose of greater accuracy the diagnosis *Brucelliasis Brucei*, bangi or traumi may be rendered once the laboratory has determined the type of organisms isolated. It is interesting to note that the pathologic-anatomic findings in men who died as a result of Brucelliasis brucei are very much like those found in cattle and caused by the brucella bangi. An educational campaign may be helpful to control the dissemination of the infection among man. Milk should be brought to boiling whenever the pasteurized product is not available, as the organism does not resist heat.

ACUTE ABDOMINAL DISEASE: REVIEW OF 1,232 CASES*

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Abdominal surgery is beginning to assume the role of preventive medicine in removing the focus before any infection has had a chance to spread to a formidable extent. But after such damage has been incurred operative measures may have only a running chance in the hazards. It is obviously recognized that there is a wide variation in the rapidity of spread of infection which constitutes a hazard always to be taken into consideration; for the advent of classical symptoms following infection becomes synonymous with more or less irreparable damage which may or may not be relieved by operation.

Fortunately the class of cases in which the infection spreads rapidly and produces active symptoms is comparatively easy of diagnosis. However, treacherous developments may occur without symptoms, and this class of cases makes the greatest contribution to the mortality rates. There is no safeguard against this treachery except operation within the first twenty-four hours, and a prompt diagnosis of such an emergency can be arrived at only through frequent observations and critical study with consultations. Furthermore, when damage has been wrought, morbidity and mortality do not cease even after recovery from late operation. Our follow-up records indicate that there is approximately a remote death rate of 1 per cent from obstruction following late operation for acute appendicitis after leaving the hospital, to say nothing of hernia and other disabling sequelae which often follow recovery from all classes of peritonitis. Inasmuch as the fate of the patient is frequently sealed after the first twenty-four hours, an early intensive study becomes of utmost importance.

The geographical incidence of surgical disease is perhaps well-nigh uniform. The records of the Harbin Hospital in a section fairly representative of 64.2 per cent of total population of the United States (1920) for a period of twenty years indicates that 45.5 per cent of total postoperative deaths from all classes of disease preceeded from the acute abdomen, chronic intraperitoneal conditions giving 8 per cent. The Mayo Clinic Bulletin reports for the year 1929, 260 operations for acute abdominal conditions (appendicitis 172, mortality 2.9 per cent), while our own records give for the same period 176 similar operations (appendicitis 143, mortality 2.1 per cent), the resident population of the respective sections being about the same. Except for the matter of local incidence of acute surgical disease the two situations could not be compared in other respects.

The laity should be educated to regard every abdominal pain as serious until it is proved to be otherwise. Surgery in all of its phases has lingered long in this stage of progress, due perhaps in part to an undue worship of the value of surgical skill and an over-ambition for an accurate clinical diagnosis which finally becomes worthless in bettering the prognosis.

Leaving out a consideration of appendicitis which rarely fails of prompt recognition, our errors in pathological diagnosis were 21 per cent, while the mistakes in recognizing a surgical condition were practically nil, for it will be discerned from the table that nearly all of these errors fell in the column for early operation. Obviously any ambition to lower that percentage by prolonging a clinical study beyond twenty-four hours would tend to create the pitfall of late operation—a potential tragedy in every acute abdomen. Our statistics, covering as they do a period of twenty years, indicate that the deadline of the acute abdomen should be drawn shortly after the end of the first twenty-four hours. To paraphrase Shakespeare:

> There is a tide in surgical disease
> Which taken early leads to recovery;
> Omitted—all is bound up in shallows and misery.

This high tide falls within the first twenty-four hours which in the absence of extraneous complications should offer 100 per cent recoveries.

In the very acute cases the medical attendant is usually driven by the activity of symptoms to an early diagnosis, but in the atypical or apparently mild cases we all fall victims to that fatal procrastinating phrase "when necessary" in applying intensive study and consultations. A difficult obstacle obtains in the protestations of patient and family, that so much "fuss and feathers" is made at times over such trivial ailments, and pending a diagnosis in the very active types of cases, that it is difficult to maintain heroic inactivity as to morphine and purgatives in the face of the

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insistency of the family. While morphine is very acceptable to the patient, it is a pernicious practice to give it and request a report the next day if the patient is not better; for nothing should be done to the patient until a diagnosis or plan of action has been decided upon. Every hypodermic injection of morphine for abdominal pain should be followed by a visit in twelve hours. The clinical picture should not be disturbed during the period of deliberation, for such treatment is no menace to non-surgical disease. If for any reason delay of radical treatment seems advisable, the perils of late application of surgery should be explained, for indecision at the end of twenty-four hours is fraught with many hazards. Acuteness of symptoms that cannot be explained away at the end of this period, where the clinical picture has not been disturbed, will usually establish the diagnosis of a surgical condition by exclusion. The use of a hypodermic injection of morphine may lead to a false belief that the trouble is of a transient nature, and the fact that morphine occasionally fails to relieve suffering aids the diagnosis of a surgical condition. Laboratory aids and x-ray examinations should be made, but negative findings should finally be subject to discount. After weighing all the evidence from a critical study, a moral obligation may arise where the surgeon must choose the lesser evil among mistakes of diagnosis, and historic evidence leads us to believe that inaction has been a greater surgical sin than overaction. One evil is operating occasionally for a lesion which might have been relieved temporarily by palliative treatment, and another is a death because of late operation.

We believe that the unwarranted death rate from late diagnosis in the acute abdomen arises more from a lack of intensive study than from any absence of peculiar diagnostic skill. It should be borne in mind that certain complications of age, obesity, disease and pregnancy add not only to the difficulty of diagnosis, but increase the hazards of the disease in question as well as that of early operation, and under these circumstances conservatism may become advisable. While it is usually not to be depended on, there is a chance for spontaneous recovery, temporarily at least, in every suspected acute abdomen under palliative treatment.

For instance, a young stenographer was treated for an attack of peritonitis for three weeks, widespread adhesions being evident. A tentative diagnosis of past rupture and hemorrhage from a Graafian follicle was made. Widespread adhesions were found with small crater in the right ovary, which was sutured. Delay of operation multiplied many times the morbidity with a prospect of future trouble, and for six months the patient was partly invalided.

In another case an unmarried teacher, 25 years old, weighing 94 pounds, suffered the same condition, which was properly diagnosed in six hours, the peritoneal cavity proving to contain a moderate amount of blood. It was thought advisable to remove the right ovary along with the appendix, and her convalescence was prompt and subsequent health has remained good since she has gained twelve pounds in three months.

The peritoneum damages itself in every successful fight with infections, however mild, and leaves extensive adhesions in so doing.

These difficulties should be explained to the family, and it requires courageous judgment on the part of the medical attendant to declare an ultimatum as to the hazards of the case, for we all are inclined to hope for the best while we fear the worst. We cannot expect an unwavering decision on the part of the patient so long as our own judgment wavers. Sir Astley Cooper's qualifications of a surgeon, though a century old, still stand and apply especially to the acute abdomen: an eagle's eye, a lion's heart and a lady's fingers; which translated into modern terminology would mean, intelligent observation, courageous judgment and refined technic. In the whole domain of surgery there is no ordeal that tries so severely the wits and courageous judgment of the surgeon as that of the acute abdomen. It is trying because a life often depends upon a prompt decision.

When operative treatment has been decided upon, the location of the incision is important, and when in doubt the right rectus type on a level with the umbilicus will furnish more options than any other. Operative procedure wisely advised and executed with standard technic within the first twenty-four hours in the absence of certain complications, offers an economical morbidity, with a theoretical mortality of zero.

Through an impelling ambition for thoroughness, operators at times may inflict an unnecessary amount of surgical trauma in establishing a negative operative diagnosis. Unnecessary evisceration may be cited as an instance. While a surgical condition may be readily recognized on opening the abdomen, its cause requires investigation at all hazards, provided operation has been applied early, for late surgery amounts to but little more than salvage. We can recall only two instances where apparently unnecessary evisceration would have saved trouble.

One was a boy, age seventeen, operated upon for acute appendicitis with no leucocytosis, with a mildly pathological appendix. Active pains continued, however, and on the third day he was operated upon again. An en-

terolith was found which had finally lodged at the ileocecal valve, and was relieved by enterotomy. He recovered.

In another case a man, age twenty-two, with a similar operative diagnosis, had persistent symptoms of obstruction, and secondary operation on the third day revealed advanced ileus of undetermined cause. He died.

Clinical records of the Harbin Hospital for a period of twenty years show that there were 1,232 acute abdomens treated by ourselves, with 384 cases operated upon within the first twenty-four hours with no mortality, while 848 were operated upon after that period with 63 deaths (7.3 per cent), not including cases of obstruction which developed in the hospital, trauma, puerperal conditions, kidneys and hernias in which the diagnosis is usually obvious. *Table 1* shows the relative incidence of various causes of acute abdominal disease in comparison with disease arising in a general surgical experience. While a discussion of the postoperative treatment does not come within the province of our title, we urge the importance of making the patient comfortable, forcing fluids by extraoral routes and take no chances on epigastric tympany, as it is the first evidence of acute dilatation of the stomach.

The most unpreventable, yet most easily cured of all abdominal disease is appendicitis, and there is no prospect that this hazard can be mitigated except by early operation. Osler says that in every death from this cause somebody is to blame. Acute appendicitis carrying a theoretical mortality of zero claims, according to our records, 40 per cent of all postoperative deaths from abdominal disease. The unwarranted economic loss from morbidity and mortality from this source is appalling, because it afflicts in the main human life at a high tide of efficiency. Fifty per cent of the total deaths from acute appendicitis occur in the 89 per cent of patients under forty

Table 1

	Operations Within 24 Hours		Operations After 24 Hours		Total Series		
Acute Abdomen From Disease	Cases	Deaths	Cases	Deaths	Cases	Deaths	Remarks
Appendicitis Drainage	336 12%	0	744 48%	31	1080 38%	31	Average duration symptoms 50 hours Mortality clean cases 0.4 per cent Mortality drainage cases, 8.0 per cent Mortality over 40 yrs. old 9. 8 pct. Mortality total 2.8 per cent
Perforation Peptic Ulcer	7	1*	8	6	15	7	All correctly diagnosed
Cholecystitis	8	0	26	8	34	8	2 diagnosed appendicitis 1 diagnosed obstruction others forced operations
Intest. obstruction contracted outside hosp.	6	0	22	10	28	10	
Salpingitis	7	0	22	0	29	0	2 diagnosed perforated ulcer 8 diagnosed appendicitis
Tubal pregnancy	3	0	8	0	11	0	
Diverticulitis Meckel's	9	0	2	2	11	2	8 diagnosed appendicitis 2 diagnosed obstruction 1 diagnosed correctly
Diverticulitis colon	0	0	7	2	7	2	
Intussusception	3	0	1	1	4	1	
Rupture abscessed mesenteric lymph node	3	0	0	0	3	0	2 diagnosed obstruction 1 diagnosed appendicitis
Ovarian cyst strangulated or rupture	1	0	5	0	6	0	
Liver abscess	0	0	2	1	2	1	
Pancreatitis hemorrhagic	0	0	1	1	1	1	
Post cesarean rupture preg. uterus	1	0	0	0	1	0	
	384	1*	848	62	1232	63(5.1%)	Errors in diagnosis 21 per cent, excluding appendicitis

*Primary recovery death 16th day from obstruction.

years old. In 1,080 cases (mortality 2.8 per cent) there were 336 (or 31 per cent) operated upon within the first twenty-four hours with no mortality. Twelve per cent of these (of the 31 per cent) were drained and 143 lived over five miles from the hospital; and our statistics show that the mortality of acute appendicitis at an average of 72 hours duration of symptoms is seven times that at 32 hours, and the treachery of acute appendicitis is further indicated by the fact that 30 per cent of these deaths were given a favorable operative prognosis. London and St. Thomas hospitals report (1920-23) 524 (or 20 per cent) of cases operated upon in the first twenty-four hours with a mortality of 1.7 per cent, while a total of 2,578 cases gave a mortality of 6.7 per cent.

The syndrome of pain, nausea and vomiting and abdominal rigidity with slight rise of temperature should be constantly borne in mind, as there is rarely a case that lacks all of these symptoms. The most constant symptom is local rigidity, though it is lacking in the first few hours, and in the post-cecal type; and where the appendix is situated in the pelvis rigidity may be observed in the left side. Early observation of a case can discern these symptoms better than any subsequent period. Leucocytosis is a more constant early phenomenon in acute appendicitis than it is in other classes of acute abdominal conditions for the reason that an acute attack is usually a culmination of pathological processes frequently symptomless and more or less chronic in type. In a consecutive series of 100 cases operated upon in the first twenty-four hours, 87 showed an average total leucocyte count of 7,500 to 14,000. Temperatures over 101° F. during this period of observation should excite suspicion of other or collateral disturbances such as pneumonia or pyelitis, still bearing in mind that appendicitis may be the primary pathological condition. At times it is wise for the surgeon to depend largely on the medical attendant's primary observation. It should be remembered that there is no necessary relation between the activity of symptoms and pathological developments. There are few conditions in younger people that simulate appendicitis, while in certain complicated cases the problem may be difficult. It is exceptional that we can ever be sure enough of a positive diagnosis of appendicitis within twenty-four hours in the presence of extremes of age, pregnancy, disease and obesity, but with close observation operation can be applied before the advent of an irrecoverable stage of the disease. The law of age incidence of appendicitis should be constantly borne in mind.

Peptic Ulcer.—Five of the 8 recoveries from perforated ulcer gave no previous history of ulcer and the simple operation of infolding with sutures, using the omentum as a patch, has been followed by clinical cures. The gravity of symptoms usually makes the diagnosis easy.

Cholecystitis.—The conception that the gall-bladder is a primary link in the subsequent chain of pathologies has enabled us to make more intelligent prognoses. Any gall-bladder operation becomes dangerous in the presence of cholangeitis and hepatitis on account of the danger from an infected gall-bladder, which, like appendicitis, carries little danger so long as the infection remains localized. Of course, as in other similar conditions, the collateral end results of infections cannot be reduced to positive cultural findings. Surgical trauma is an affront to all acute spreading infections, especially to the end results of an infected gall-bladder. The same thing is true in peritonitis, but in a lesser degree. The morbidity from a diseased gall-bladder constitutes a serious problem in itself. In the gall-bladder syndrome we have furnished one of the most definite clinical pictures that needs no discussion.

These considerations lead us to believe that it is good surgery to do a gall-bladder operation during the first twenty-four hours of the acute attack when it rises to the gravity of symptoms of the acute abdomen, because its severity constitutes a serious threat for a forced operation later. In 8 operations in the first twenty-four hours with no mortality, 2 were diagnosed as appendicitis, 1 as obstruction in a patient who had had an abscessed appendix five years previously. These three patients were young, stout multiparae, and five others were operated upon because of the acuteness of their symptoms. The remaining 26 cases (mortality 34 per cent) were forced operations where palliation had failed (concurrent series, acute and chronic cholecystitis 199, deaths 12). Results would probably have been better had all of these more or less advanced cases been submitted to immediate operation when admitted to the hospital. In the absence of accurate data it is our belief that there were not more than 20 per cent of this total number of acute cases concurrent with this series that yielded to palliative treatment, for no case died in the hospital without operation. The ingestion of quantities of fluids with bicarbonate of soda with no food whatever seems to be the best form of palliative treatment. Better internal medicine is tending to reduce the number of surgical abdomens except appendicitis. Especially cholecystitis is being reduced.

Ileus.—Acute intestinal obstruction when complete usually offers little difficulty in diagnosis, and the 28 cases listed originated outside the hospital. Obstruction is often a terminal complication of postoperative peritonitis. Our statistics show that the rate for secondary operations for obstruction following acute appendicitis while in the hospital were 0.4 per cent for clean and 0.6 per cent for drainage cases. Two per cent has been given as the average. The mortality rate for cases of obstruction operated upon after twenty-four hours was 45 per cent as listed.

Salpingitis.—Acute salpingitis is usually referred to by gynecologists as a condition easy of diagnosis, but our errors in this condition were greater than in any other acute abdominal condition, as two very acute cases were tentatively diagnosed perforated ulcer and eight as appendicitis in a total of 28 cases. There is no usual condition in the abdomen that inflicts such a painful and well-nigh interminable morbidity as that of very acute salpingitis, and restoration of function in these violent types is not to be expected. One death from appendicitis was attributed to the fact that the case was treated palliatively as salpingitis. Of course, operating for a very acute gonorrheal salpingitis on the third or fourth day spells disaster, and perhaps every surgeon who has operated after subsidence of symptoms in these cases wishes that he could have operated in the first twenty-four hours. However, the great majority of cases of acute salpingitis do not rise to the gravity of symptoms of the acute abdomen and should be treated with palliative measures in the hope of restoration of function.

Tubal pregnancies and strangulation of cysts are fulminating in the type of their symptoms in the minority of cases and are usually correctly diagnosed. The same is true of diverticulitis of the colon.

The 11 cases of Meckel's diverticulitis were diagnosed in 8 instances as appendicitis, in 2 as obstruction, and one was correctly diagnosed.

Intussusception is only exceptionally missed in the diagnosis.

Ruptured Abscess of Mesenteric Lymph Nodes.—The operative diagnosis of this condition was made in 3 cases by exclusion. Two were diagnosed as obstruction and one as appendicitis. These cases were very fulminating in type, reaching the operating table within the first twenty-four hours.

One was a pupil nurse who had had an operation for sub-acute appendicitis seven days previously, at which time the abdominal cavity was surveyed. There was primarily noted an enlarged lymph node in the mesentery of the terminal ileum, and in this area was noted evidence of a spill of infection along with a disappearance of this node at second operation one week later. The inverted stump of appendix was negative in appearance.

Another similar case in a multipara 34 years old received a preoperative diagnosis of obstruction. The appendix was negative and the alimentary tract was thoroughly inspected with negative findings.

The case of an undernourished boy five years old was diagnosed as appendicitis and a well protected pathological appendix, post-cecal, was dug out. The largest of a chain of mesenteric lymph nodes was excised and pronounced adenitis. Three months later this patient was operated upon for ventral hernia and the chain of lymph nodes was noted as before. All of these cases showed that characteristic spill of infection that could not otherwise be accounted for. For drainage cases their convalescence was unusually prompt.

SUMMARY

(1) About half (45.5 per cent) of postoperative deaths from miscellaneous diseases generally proceed from acute surgical conditions within the abdomen.

(2) As a working basis the suspected acute abdomen should constitute a challenge to the wits of the medical attendant and surgeon to make a diagnosis within the first twenty-four hours or be prepared to explain the hazards of such delay.

(3) The laity should be taught to regard every abdominal pain as serious until it is proved otherwise.

(4) Failure of diagnosis of a surgical condition in the first twenty-four hours is due more to a lack of frequent observation and consultations than to absence of a peculiar ability.

(5) Waiting for classical symptoms is the pitfall of many a conscientious endeavor that fails to better the prognosis, and symptoms that cannot be explained away at the end of twenty-four hours will usually establish the diagnosis of a surgical condition.

(6) Negative laboratory and x-ray findings should in the final analysis be subject to discount.

(7) The complications of age, disease, pregnancy and obesity constitute a serious obstacle both to accurate diagnosis and safe surgery in which the choice of hazards must be made.

(8) Historic evidence leads us to believe that inaction has been a greater surgical sin than overaction, and surgical progress has lingered long in the unavoidable stage of wisely choosing evils.

(9) Difficult as it may be, nothing should be

done to the patient so long as the diagnosis is being deliberated.

(10) Fifty-six per cent of postoperative deaths from all diseases proceed from intraperitoneal causes, and in 384 acute cases of the latter there was no mortality following operation within the first twenty-four hours.

(11) Excluding appendicitis, practically all of the 21 per cent of errors of preoperative diagnosis fell in the column for early operation.

(12) In 336 cases of acute appendicitis that had operation within the first twenty-four hours, 143 lived over five miles from the hospital, 12 per cent having been drained and 87 per cent showed leucocytosis, mortality of total series being 2.8 per cent.

(13) Three cases of acute cholecystitis and ten of acute salpingitis operated upon early through error of diagnosis proved to be good surgery, and raised the question of revisions of rules in such cases.

(14) The mortality in 1,232 cases was 5.1 per cent, and 50 per cent of deaths proceeded from the appendix.

(15) In the absence of extraneous complications may it not be wise for the sake of morbidity as well as mortality to operate for every surgical disease within the abdomen that suddenly attains the gravity of the symptoms of the acute abdomen, provided such operation is applied within the first twenty-four hours?

DISCUSSION (*Abstract*)

Dr. Frank K. Boland, Atlanta, Ga.—I notice that Dr. Harbin had no cases of intestinal obstruction originating within the hospital. All of his cases were contracted outside of the hospital. I congratulate him on that, as I do on the other figures here. I think 384 cases of acute abdominal disease, operated upon with no deaths, within twenty-four hours, are certainly very fine statistics.

In our experience the commonest cause of postoperative obstruction is the failure to peritonealize raw surfaces.

We are prone to criticize our medical friends sometimes for not referring these cases sooner for operation. I believe we can forgive them for not making a diagnosis of the acute abdomen, but not for giving castor oil and morphine. That is an unpardonable sin.

I should like to speak a word in behalf of the man who sees one of these cases first. It is much easier, I believe, as a rule, for the surgeon who comes on the scene twenty-four or forty-eight hours after the inauguration of symptoms to make a diagnosis and decide that an operation is imperative than it is for a medical man who sees the case from a jump.

We must not wait for all the so-called classical symptoms. We have to operate in the absence of a great many symptoms.

The only constant symptom of acute intestinal obstruction is pain. When a patient has severe persistent abdominal pain, we should base our diagnosis of acute intestinal obstruction on that. When we can give these cases more prompt treatment, we will do a great deal toward diminishing the cost of medical care. We will cut down expenses in hospitals and also relieve sorrow and tear-shedding at home.

Dr. G. V. Brindley, Temple, Tex.—Next in importance to the manner in which the operation is to be performed is the time at which it is to be done. Frequently the time element is a more vital factor in the determining of the end result than the experience and skill of the surgeon. These statistics tell us with what safety abdominal surgery can be performed when it is done skillfully and at the proper time.

Dr. Harbin has used twenty-four hours as the dead line between early and late operations. I do not know how a better classification can be made. However, I know that Dr. Harbin will urge that many of these patients be operated upon at the earliest possible hour. An operation performed twenty-four hours after the onset of acute symptoms is sometimes a late operation. The fact that 12 per cent of the patients with acute appendicitis operated upon within twenty-four hours were drained substantiates this statement. The acute abdomen is an emergency condition and Dr. Harbin has shown us that it demands immediate operation, whether it be 2 o'clock a. m. or two o'clock p. m.

Particularly would I stress the urgency of early operation in acute perforated peptic ulcer and acute intestinal obstruction. The diagnosis of these conditions usually can be made readily and these patients should be operated upon within a very few hours of the onset of their acute illness. When we refer to the statistics and see that there was a mortality of 75 per cent for the patients with acute perforated peptic ulcer and a mortality of more than 40 per cent for acute intestinal obstruction when these patients were operated upon late, then we can appreciate the need for operating at the earliest possible hour.

I would raise one question for consideration, that is this: should the patient with acute cholecystitis be operated upon within the first twenty-four hours? We know that acute gall-bladders seldom perforate and also that the acute symptoms will usually subside. It has been our custom to handle patients with acute gall-bladder disease expectantly for at least a short time.

Dr. Horace Reed, Oklahoma City, Okla.—My discussion will be limited to the question of acute appendicitis. It may not be exactly orthodox.

I have about reached the conclusion that if we are to make progress in lessening the mortality from appendicitis we should select our propagandists from among those who have had it. With doleful voices we lament our apparently increasing mortality and lay the blame on the patient who takes castor oil or other cathartic.

An incident of sixteen years ago awakened me, and since that time I have asked each of my patients having a so-called fulminating type of appendicitis what he or she thought the trouble was at the onset. The replies have been almost without exception, "stomach trouble" or "indigestion." The first sign of acute inflammation of the appendix is pain in the "stomach," and this pain is not different from the impression we carry of the belly-aches of childhood.

The patient is always present when the first symptom arrives. If he has not concluded from the location of

the pain that he has "indigestion," or "stomach trouble," he gets strong support for such conclusion when he vomits what he assumes to be the particular substance he ate, and which he suspected of causing the trouble. When, like Will Rogers, we read the papers, we learn that a well-known prize fight promoter, sojourning for awhile in Florida, has indigestion, and he is conscious of having over-indulged in rich food. His stomach ache was a natural consequence. He waved aside the suggestion that he should have a physician or go to a hospital. He knew what he had. But a few days later something happened inside, then he went to the hospital, where, according to the papers, he died following an operation for appendicitis. And we read that a member of the President's Cabinet had JUST indigestion, in spite of which he continued to go to his office. Also the papers told us about the captain of a major baseball team, who would not let an attack of indigestion take him from his team-mates, who were struggling to get into first place as the end of the season approached. But the cabinet member and the captain both joined the prize fight promoter because each knew what his trouble was and acted accordingly.

If we are to lower the mortality, we must get to the patient before the attack of appendicitis. We must educate him. But before we undertake to give out information let us make sure that we know what we are talking about. I have never been more chagrined than when I read in a health magazine, which is sponsored by one of the greatest medical associations in the world, an article on acute appendicitis. This article, in describing the symptoms of acute appendicitis, properly states that pain is the first symptom and is invariably present. It then goes on to state that the location is "usually in the right lower quarter of the abdomen." Shades of Murphy arouse and protect us from such misinformation!

Not until the public has been properly instructed, perhaps through some public health organization, in the early signs and proper treatment of acute appendicitis, will there be any appreciable improvement in the mortality and morbidity.

Dr. A. C. Jackson, Jasper, Ala.—In making the annual death report in a small hospital where I do my surgery, we found that 36 patients died within the first twenty-four hours after admission, 6 within the next twenty-four hours, and 14 died after being in the hospital more than forty-eight hours. In other words, 75 per cent of these deaths were in cases unloaded at the hospital door in such a moribund condition that they died within forty-eight hours; 25 per cent lived for a longer period and could correctly be charged up as institutional deaths.

A few years ago I reported a series of perforated gastric and duodenal ulcers before the State Association of Alabama, in which 12 cases were operated upon. Of these 12 cases, 8 were operated upon within the first eight hours after the perforation occurred, and all of these recovered; of the 4 who waited longer than eight hours for operation, one recovered and three died. Most of you have had experiences similar to this.

The older men who have done pioneer work in the practice of medicine should not be censured, but in our contacts with them we should educate them to the fact that immediate operation in all acute abdominal conditions is far more urgent than a correct diagnosis.

Perforated peptic ulcer has been quite prevalent in our section for the past few years, and so we have discussed this subject before our staff meetings at the hospital and the County Medical Society with the fine result that we are now getting some of these cases for operation within four hours after perforation occurs, and others within six and eight hours. I believe that the education of the men in general practice and the laity will do more than any other one thing to lower our mortality rate.

Dr. Hubert A. Royster, Raleigh, N. C.—Just three years ago I was taken off the train at 3 o'clock in the morning at Baltimore on my way North to put the finishing touch on my book on appendicitis, and I called Dr. Finney and had my appendix removed. As I looked back, I had had stomach trouble for two years, and I had a colic three nights before and could hardly get off the train. In appendicitis, the attack is not the disease, but only the knock at the door. It is a continuing process, a going concern, and once appendicitis always appendicitis.

Let me call your attention to the two factors in mortality which go behind the returns, procrastination and purgatives, the twin factors of fatality in appendicitis. A campaign has been started in Philadelphia, and we are starting one in North Carolina soon on this basis. Druggists are asked to put placards in their stores: "Do not take purgatives in abdominal pain. See your doctor, first."

Then, I say, let us get to the physician before the patient sees him, because the general tendency not only among the laity, but among physicians and surgeons, is to give a purgative for driving something out of the intestinal canal that has nothing to do with the case, like "the flowers that bloom in the spring."

We have relegated this most frequent of acute abdominal diseases to the shelf of solved problems, and are giving ourselves the feeling that we are interested in more modern and, forsooth, more scientific matters.

The mortality from appendicitis is steadily increasing. In Philadelphia, between 1913 and 1923, the mortality increased 18 per cent. Out of the first year's check-up it was found that 207 deaths were due, practically entirely, to purgatives. Seventeen thousand cases died in 1929 in the United States, and of those 12,000 had purgatives, and 11,000 of those probably died as the result of purgatives. What are we going to do about it? The fatal thing is that most physicians consider that, when they send their patients to their favorite surgeon, he is brilliant and skillful enough to save them even at the eleventh hour.

When an unruptured appendix comes to the surgeon, the physician is responsible for the recovery. When the appendix is gangrenous or perforated and the patient dies, the blood is on the head of somebody who has blundered. If he gets well, it is to the credit of the surgeon.

Dr. J. G. Gaither, Hopkinsville, Ky.—I wish to say a few words along the same line in which the paper is directed; that is, whether or not we have been improving in the rapidity with which these patients are brought to the table. A series which I have studied consisted of 684 cases of acute, violent appendicitis from 1920 through 1929 inclusive, making a decade. There were of the 684 cases, 175 which had drainage tubes placed in them. One in every four had to have a drainage tube in the abdomen. There were the same number of patients proportionately in 1920 who had to have tubes placed in the abdomen as there were in 1929. In other words, in ten years, in my own community, there was

no improvement in the handling of acute appendicitis, either through the fault of the physician who saw them or in the knowledge of the laity themselves as to what should be done. So, I think it is certainly true that the handling of acute appendicitis still leaves much to be desired.

Dr. Harbin (closing).—Dr. Boland in discussing our paper indicated that there was no evidence in it to indicate that we have postoperative intestinal obstruction. We do have cases of obstruction following operation, and in the paper just read the death rate for the obstruction cases has been given for the patients that incur obstruction before reaching the hospital. As stated in the context, cases of the acute abdomen from trauma, hernia, puerperal causes and postoperative obstruction developing in the hospital, were not included in this study for obvious reasons.

MIXED TUMOR INVOLVING HARD AND SOFT PALATE: CASE REPORT*

By ALFRED BLUMBERG, M.D.,
and
GEO. H. B. TERRY, M.D.,
Oteen, N. C.

Tumors are not common in the palate. There have been reported, however, about 100 mixed tumors in this region, of which, according to Sonnenschein, less than fifty were found in the soft palate. These mixed tumors are neoplasms composed of several kinds of tissue elements. G. B. New states that this type of tumor presents a hard, nodular surface with the feel of malignant growth. Its location is usually lateral and Edward H. Hatton describes the tumor as being unilateral, slow growing, stimulated to growth at the time of puberty or afterwards.

On histologic examination the tumor elements are of epithelial and connective tissue origin. Morant Baker removed a tumor from the upper surface of the soft palate of a child three years old. The tumor was exhibited at the Pathological Society of London in April, 1881, by Dr. Hale White, who described the growth as being two inches in length, with a wide base and consisting of epidermis, papillae, corium and fat, and having on one surface a plate of cartilage. This description may be applied to many of the tumors removed since by other surgeons. The divergence from this description arose as a matter of opinion, inasmuch as some of the investigators considered certain elements as being of endothelial (Volkmann, Eisenmenger, etc.) origin rather than epithelial (Krompecher, Coenen, Clairmont and others). The microscopic findings in one of Sonnenschein's patients show the presence of deeply stained epithelial cells invading mucoid or hyaline stroma; squamous epithelial masses, with cornification and calcification; connective tissue stroma which in places was hyaline, apparently compressing epithelial strands and mucoid and cartilaginous areas. Fat cells were occasionally found and present in small groups. Blood vessels were not numerous. The tumor of his other patient contained chiefly mesothelial elements, including connective tissue and large areas of cartilage. In addition there were bands of large, lightly stained epithelial cells and many alveoli lined by cuboidal and flat epithelium. Acidophilic homogeneous material, apparently colloid in character, was found within the alveoli. It may be noticed that cartilage is an element usually present. An exception is G. V. Boyko's patient, in whom a tumor was found which showed nodules and cords of round and oval epithelial cells in an abundant connective tissue framework, which at times proliferated to form distinct bands. Where mucoid degeneration existed, the epithelial cells were swollen and the nuclei were crowded toward the periphery of the cell. In other places these cells had an arrangement suggestive of gland forms. There were no mitotic figures. Blood vessels were abundant and comparatively large. Boyko fails to mention the presence of cartilage.

Almost every writer on the subject of mixed tumors prefers to call attention to the possibility that these tumors may become malignant. This warning is chiefly due to the microscopic description of the pathologist who finds elements of the kind which give the impression that the new growth belongs to the malignant variety. In conclusion, after having described the details found on microscopic examination, he will render a diagnosis of complex terminology. In the past, terms such as enchondroendothelioma, myxochondro-endothelioma or myxochondrocarcinoma were not unusual. Other terms were those of cylindroma, basal cell carcinoma and endothelioma. These latter terms were most likely applied to tumors free from cartilage. Apparently the tumors removed from the palate and described as being sarcoma belonged also to the class of neoplasms of clinically benign character, for they produced no metastasis and their removal was followed by permanent cure.

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*From the United States Veterans' Hospital No. 60, Oteen, N. C.

Christopher Heath mentions a woman 48 years of age who had a tumor of the hard palate the size of a horse chestnut, which had been noticed from childhood, but had latterly increased rapidly. He succeeded in removing the growth, which proved to be a round cell sarcoma, and by the application of the actual cautery to the bone from which it sprang he induced a superficial exfoliation and a permanent cure.

S. Lodge and W. O. Lodge give an account of "Recovery from Tubal Deafness Caused by Sarcoma," in which they state that they removed the tumor from the soft palate. As a result, the pressure upon the Eustachian tube being relieved, the unilateral deafness of eight years' standing disappeared.

It is inconceivable that a sarcoma should fail to give rise to metastasis after such long periods of existence as mentioned in these two instances. New considers that mixed tumors are potentially malignant and that when they recur after a primary complete removal they metastasize. Apparently, however, this refers to mixed tumors in other locations, for he describes three palatine tumors none of which showed such metastasis, though at least one of them had to be cauterized twice, and in two instances the tumors ruptured in the removal. Boyko considers these growths benign, but warns that they may become malignant. V. P. Blair claims that, in his experience, these growths are apparently almost always benign, and only occasionally after a prolonged course do they show very marked malignancy. It is to be regretted that Blair fails to give a description of an actual instance in which such metastasis occurred, provided the primary tumor was in the palate.

With the evidence on hand one may safely say that mixed tumors belong to the clinically benign variety of growths and if they ever cause symptoms it is due to their size and location, as under certain conditions they may give rise to complications. It has been pointed out repeatedly by the various authors that bone atrophy and perforation of the hard palate may occur as a result of pressure. Difficulty in deglutition and perhaps in respiration may occur if the tumor is extensive in size or so situated as to give rise to such symptoms. New found that one of his patients had a palatine mixed tumor which involved the antrum. If metastasis with consequent fatal termination occurred in any instance in which palatine mixed tumor was the first focus of a new growth it has escaped our attention.

Our impression is that in their behavior these growths can be compared with the giant cell tumors of bone. Microscopically their appearance alarmingly suggests malignancy. Clinically once the tumor is completely eradicated, no further activity is to be feared. These growths are most probably embryonic in origin. Fraser believed that mixed tumors may be the end result of poor drainage of gland ducts. He does not refer, however, to palatine tumors. Hatton believes that these tumors arise from embryonic rests, the cells are epithelial in origin, and the structures are stimulated to growth during puberty. To this Morant Baker's and Christopher Heath's patients are exceptions, as in the former instance the patient was three years old and in the latter case the tumor made its appearance during childhood. Wood, Eggert and others also are in favor of the embryonic theory. A question may be raised of whether low grade chronic inflammations may induce such cell proliferation as is seen in these tumors. Bearing in mind the fact that healed tuberculous lesions do show the presence of connective tissue, cartilage, calcification and sometimes bone structures, and that similar combinations of connective tissue, cartilage and bone are found in tonsils in which the inflammation was of long standing and intermittent, one could be inclined to give a thought to the "inflammatory theory." For the time being, however, the cell rest theory is perhaps the most correct and with the acceptance of this theory the terminology used in diagnosticating these neoplasms should be "mixed tumor."

The treatment is surgical.

The following case history describes our findings:

The patient, a white male, aged thirty-five years, reported for examination on March 26, 1930. He complained of hoarseness and sore throat, which he attributed to an attack of influenza during his army service about twelve years previously. He claimed that since then attacks of sore throat had occurred at varying intervals and persisted for some time.

His history is manifestly unreliable. He is of subnormal intelligence and seems obsessed with the idea of connecting his present disability with his army service. He claims that at one time his occupation was that of a steam shovel fireman. At present he is a farmer. His mother, two brothers and three half-sisters are living and well. His father died some time ago, but his age and the cause of his death are unknown. The wife of the patient is not in good health and their three children are living and well. He denies the use of alcohol and denies venereal history. At one time he asserted that his throat trouble dated from the time that he was gassed during his army service.

Examination of the nose revealed no abnormality. The sinuses were clear, both by transillumination and by x-ray. He had a chronic tonsillitis and also a chronic non-suppurative otitis media, with hearing for spoken voice reduced to 3 inches for the right ear and 5 feet for the left. On the right side of the roof of the mouth was seen a large swelling, apparently soft and fluctuating. This was thought at first to be a cyst

or abscess, and two days later under 0.5 per cent novocaine anesthesia an attempt was made to aspirate the contents with a large needle. Nothing was obtained by aspiration. An incision was then made down to the tumor mass, which was found to be egg-shaped and encapsulated, being situated beneath the mucous membrane of the hard and soft palate. The superficial portion of the tumor mass had softened considerably, while the deeper portion had not. Enucleation within the capsule was accomplished without difficulty, the wound was partially closed with suture and no packing was employed. Three per cent tincture of iodine was applied to the wound daily and healing was practically completed in two weeks. The tumor was considered to be clinically of benign nature. On August 4, 1930, the patient reported for re-examination. There was not the slightest evidence of recurrence of the tumor locally or by metastasis. He, however, stated that his right ear had been very painful and that his deafness on that side had become much worse since the operation. He claimed to hear the voice at no greater distance than ½ inch. Examination of the ear disclosed nothing to account for the pain and a few simple tests for malingering proved that he grossly exaggerated his hearing defect.

The tumor itself was 4¼ cm. long and 4 cm. wide at its greatest dimension. It was egg-shaped, somewhat reddish in color, in places soft to touch, showing definite mucoid changes in these areas. The narrowest end had a torn appearance. The growth was well encapsulated, its cut surface somewhat mottled in appearance. In spots a milky bluish appearance was noticeable and such areas were glistening. Fine, fibrous bands were seen in different places. Microscopic examination revealed epithelial tissue, hyaline connective tissue, mucous deposit and cartilage in addition to round cell infiltration. The epithelial tissue was either deposited in groups which did not merge with the stroma or showed a lace-like arrangement. The cells stained deeply and suggested the basal cell variety. Here and there alveolar formation was distinct and the lumen of the alveoli frequently contained colloid-like material, while the surrounding epithelium was distinctly of the glandular type. Squamous epithelium was found here and there, and there was a tendency to cornification and calcification. In some areas there was grouping into nests of deeply staining epithelial cells, the nests being separated by a fine stroma. No mitotic figures were found. In some places epithelial cells were arranged into long strands and columns. The connective tissue in places was very narrow, while in other places hyaline changes were present. The cartilage was surrounded by epithelial tissue. The round cell infiltration was either gently diffuse or collected into foci. The diagnosis was mixed tumor of the palate.

The pathologist's description of this tumor is quite similar to the ones described as myxochondro-carcinoma. We refrained from using this term, as it might have suggested that the growth was malignant. On the basis of our analysis of our own case history and the ones found in the literature, it is our purpose to call attention to the fact that no absolute evidence has been offered to the effect that palatine mixed tumors metastasize or are malignant. They may recur after having once been removed, but their recurrence is not associated with dissemination of tumor tissue into other organs.

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DISCUSSION (*Abstract*)

Dr. John A. Lanford, New Orleans, La.—The present treatment of mixed tumors of the parotid type, whether they occur on the palate, the face, or other portions of the head, is becoming more efficient. The older text books tell us that mixed tumors of the palate should always be considered malignant and, although the observations of the recent writers have been different, most pathologists and surgeons still consider them potentially malignant. The recent literature is rather full of reports of mixed tumors, rather more in the last two or three years than formerly, and most of the writers still look upon them as being malignant. The reason for this view is that these neoplasms possess certain infiltrative tendencies and are very prone to recur. MacFarland recently stated that no case of mixed tumor should be considered cured just because there has been no recurrence in a few years, as there are recorded instances of recurrences, where ten, twenty or thirty years have elapsed since the primary removal of the neoplasm. My experience does not go back so far, but I have observed that they are quite prone to recur.

Mixed tumors are not seen so often in the palate as in other parts of the head. I have personally seen only one in the soft palate and one on the uvula and each of these patients recovered following complete surgical removal. However, I saw one case in which the mixed tumor was located in the trachea and while this was completely removed there was a recurrence and ultimately the patient died. This death was not due to metastases, but to extension from the primary lesion in the trachea which ultimately involved a great portion of the bronchial tree. A mixed tumor in this location would be called malignant by location.

We must not lose sight of the fact that a certain proportion of mixed tumors of the parotid type may possibly show a so-called malignant degeneration, but it is doubtful that a malignant neoplasm will spring from the cells of a benign tumor any more frequently than from the cells of any other tissue in the body. It has been my observation that relatively few mixed tumors of the parotid type show malignant tendencies, but since we recognize this possibility the location itself of such a mixed tumor in the palate should not have any tendencies deterrent to a malignant change. While I

have never seen such an occurrence and the literature does not carry a record of such a malignant tumor of the palate, the possibility should always be kept in mind.

Since a mixed tumor is made up of two distinct types of cellular elements, epiblastic and mesoblastic, it is possible for two distinct types of malignancy to develop. My observation is that the epithelial element is the one which most frequently shows malignant tendencies.

In treating these new growths radium has been quite beneficial and should be used where it is impossible completely to remove the growth by wide dissection.

Dr. Blumberg (closing).—It would be a grave error to state positively that the type of neoplasm under discussion is absolutely benign. Our contention is to the effect that no evidence exists that mixed tumors have ever been proven to be malignant when they occurred in either or both hard and soft palate, and recurrence was due to incomplete removal. The case histories quoted and the slow growing nature of this tumor corroborate our assumption. In fact, on discovering such a neoplasm one can describe its course to the patient before the history is taken. Dr. Lanford states that the recent literature quotes many instances of this type of tumors. He of course refers to mixed tumors in general. The ones occurring in the palate have been mentioned in slightly over 100 instances and their occurrence could not be a frequent one, otherwise the dental profession, having frequent opportunity to examine mouths, would have reported these tumors.

FIELD STUDIES OF HUMAN INTESTINAL PARASITES IN TENNESSEE*

By A. E. KELLER, M.D.,
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The studies which are reported at this time represent investigations of the incidence, intensity and epidemiology of the common human intestinal parasites in Tennessee. The work was begun July 1, 1929, as a joint study by the Tennessee State Department of Public Health, the Department of Preventive Medicine of the Vanderbilt University School of Medicine and the Department of Helminthology of the Johns Hopkins School of Hygiene. Up to October 1, 1930, the study of fifty-two counties had been completed. There has been a total of 25,192 examinations made on rural people. These people live in areas which are almost entirely unsanitated. The areas studied in each county were selected in order that accurate information could be obtained from the population groups most likely to harbor intestinal parasites. As far as possible, the examinations were made in family groups.

About 4,000 of the examinations were made on negroes in West and Middle Tennessee. The results, however, will be reported for the study as a whole and not according to race. Racial differences, as far as it has been determined by this investigation, are not important in a study of this kind except perhaps in the case of hookworm, in which condition it is stated that the incidence of hookworm in the negro race, under conditions suitable for transmission, is not as high as in the white race. As there are practically no negroes living in the sections of Tennessee where hookworm is most prevalent, the racial question at this time can be disregarded. It has been shown by Otto, Cort and Spindler (1929) that the incidence of Ascaris in negroes would parallel that in the white race provided both races lived under conditions suitable for the transmission of Ascaris.

The study, through October 1, 1930, represents samples from 55.6 per cent of the rural population of the State. Specimens have been examined from 2.1 per cent of the above population. This represents in the main the farm population, the group in which the parasitic problem centers.

Approximately the same number of examinations have been made on males and females in this study. No significant differences in the sex distribution of these parasites have been noted.

The technic employed in the examination of all specimens in this study was that described by Stoll (1923) and later modified by Stoll and Hausheer (1926). This method is one by which the number of eggs per cubic centimeter of feces can be determined for the different helminths. It is based upon the dilution of feces with tenth-normal solution of sodium hydroxide in the proportion of one gram of feces to 15 c. c. of sodium hydroxide solution. After this dilution is made, all of the ova in 0.075 c. c. are counted. By using this technic it has been possible to determine not only the incidence but also the intensity or worm burden of the persons infected with the different helminths. In the case of hookworm, this method differentiates between light and heavy infestations and is considered at the present time to be the most satisfactory method available for the determination of the importance of hookworm in an area.

The method of examination in the present investigation is considered to be more accurate

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*From the Tennessee State Department of Public Health and the Department of Preventive Medicine and Public Health, Vanderbilt University.

than the smear method, particularly in the case of hookworm. It has been shown by Herrick and Hausheer (1926) that the smear technic is not an accurate method for specimens containing less than 500 hookworm eggs per gram of feces. It has also been shown by Herrick, Hausheer and Pearce (1926) that the Stoll technic is accurate for specimens which contain as small a number as 200 hookworm eggs per gram of feces.

In order to determine what progress has been made in the control of intestinal parasites in Tennessee during the past fifteen years, the data compiled during 1910 to 1914 by the Rockefeller Sanitary Commission in the State will be used for comparative purposes. The method of examination used in that study was in the main a simple smear technic. It seems probable, therefore, that if the present series of examinations had been made by the smear method, the incidence comparison for hookworm would show greater reduction than those given below.

HOOKWORM

Of the 25,192 specimens examined, 1,870 were found positive for hookworm ova, making an incidence of hookworm infestation of 7.4 per cent for the entire group. When the present incidence of hookworm is compared with the incidence of 25.4 per cent found by the Rockefeller Sanitary Commission, it is evident that there has been a marked reduction in the incidence of hookworm during the past fifteen years.

Of the fifty-two counties studied to date, hookworm has been found in forty-three. In only four of these has there been found an incidence of more than 10 per cent, Fentress showing an incidence of 34.9 per cent; Cumberland, 20.7 per cent; White, 14.6 per cent; Morgan, 13.2 per cent. Fifty-six and three-tenths per cent of all the cases of hookworm found to date are localized in these four counties. There are seven counties, Claiborne, Clay, Overton, Pickett, Rhea, Scott and Roane, which are contiguous to the above counties. In each of these counties the incidence is between 5 and 10 per cent, and 23.2 per cent of all the cases of hookworm are localized there. Thus, 79.5 per cent of all the cases that have been found are confined to the above eleven counties. All of these counties are situated on or contiguous to the Cumberland Plateau. This area of Tennessee is one of the three soil provinces in which the hookworm problem was shown to be of major public health importance by Rickard and Kerr in 1926. Only 6.9 per cent of the rural population of Tennessee resides in this area, where 79.5 per cent of the cases have been found. *Fig. 1* gives a comparison of the inci-

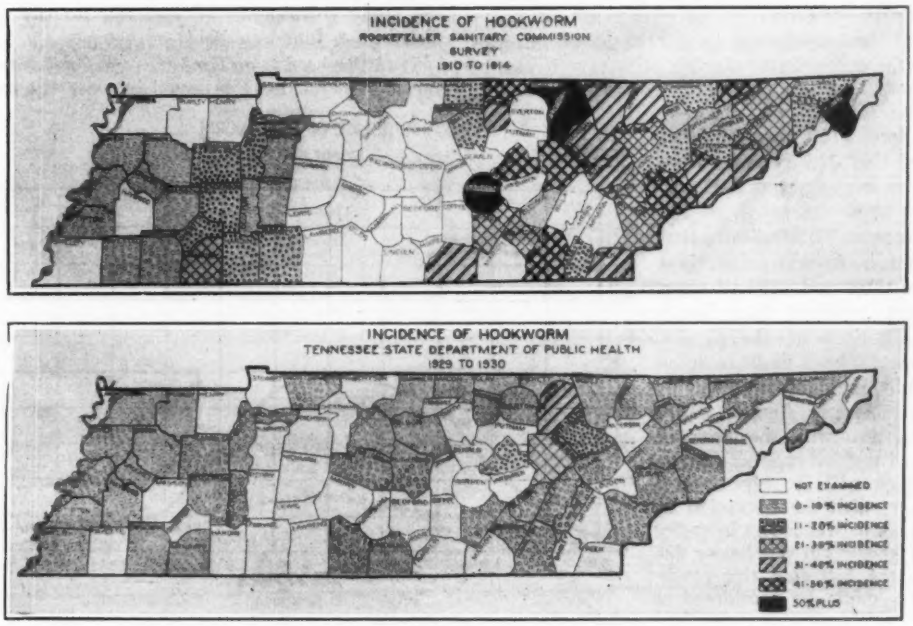


Fig. 1

Table 1

ANALYSIS OF INTENSITY OF HOOKWORM INFESTATION

Intensity	Eggs Per 0.01 c.c. Feces	Approximate Number of Worms	Number of Positive Cases	Per Cent Positive Cases
Very Light	1-6	1-25	662	40.9
Light	7-25	26-100	675	36.3
Moderate	26-125	101-500	340	18.3
Heavy	126 and over	501 and over	73	4.4
		Total	1,870	99.9

dence and distribution of hookworm in Tennessee during the years 1910-1914 and 1929-1930. From these maps, the localization of the problem in the State at the present time can also be seen.

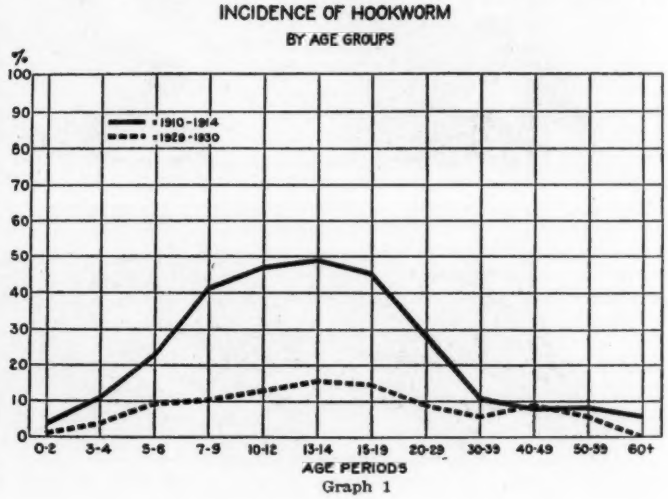
The worm burden or intensity of infestation can be analyzed by computing the number of eggs per 0.01 c. c. of feces. Using this as a basis for intensity studies, it is possible to differentiate light infestations from heavy ones which are more likely to produce clinical symptoms. At best, any correlation of the worm burden from the number of eggs per 0.01 c. c. of feces is inaccurate, but the approximate number of worms can be computed by multiplying the number of eggs per 0.01 c. c. of feces by four. *Table 1* has been arranged to show the worm burden or intensity of infestation of 1,870 positive cases.

If the intensity groups are considered in terms of the approximate number of worms, it can be shown that 77.2 per cent of the cases have from 1 to 25 eggs per 0.01 c. c. of feces (100 worms or less) and that 22.7 per cent have 26 or more eggs per 0.01 c. c. of feces (more than 100 worms). This indicates that light hookworm infestations are three and one-half times more common than heavy infestations at the present time. These findings agree with the recent work of Chandler in India (1929) and also with the work of Cort and his collaborators in China (1926), who showed that, with the exception of areas in which the intensity of hookworm was heavy due to the fact that this condition might be considered as an occupational disease in the mulberry growers in China, the number of light cases of hookworm infestation was very much greater than heavy cases. The relative proportion of light and heavy cases found by Chandler and Cort is about the same as the data from Tennessee would indicate.

Information concerning the age incidence and intensity of infestation for this series of examinations is available for only thirty-two of the fifty-two counties studied. Most of the cases of hookworm, however, have been found in this group of counties. The data compiled from these thirty-two counties will be used and comparisons will be made with the same counties studied by the Rockefeller Sanitary Commission in 1910-1914.

The age distribution of hookworm in the present study corresponds very closely to the age distribution found in 1914.

It can be seen from *Graph 1* and *Table 2* that the highest incidence is found between the ages



Graph 1

Table 2

INCIDENCE OF HOOKWORM BY AGE GROUPS

	Rockefeller Sanitary Commission 1910-1914			Present Study 1929-1930		
Age Groups	Total Exam.	No. Pos.	% Pos.	Total Exam.	No. Pos.	% Pos.
0- 2	895	31	3.4	714	8	1.1
3- 4	1887	204	10.8	1118	41	3.6
5- 6	2621	605	23.1	1917	147	7.6
7- 9	4718	1953	45.7	4469	456	10.2
10-12	4884	2298	47.0	3867	504	13.0
13-14	2868	1407	49.0	1723	270	15.6
15-19	5183	2341	45.1	1122	164	14.6
20-29	5513	1550	28.0	201	18	8.9
30-39	4820	567	11.7	184	10	0.6
40-49	3042	250	8.2	114	10	0.7
50-59	1552	139	8.9	53	3	0.5
60 and over	945	53	5.6	37	0	0.0

of five and nineteen, the school age groups. In this series of examinations, 94.4 per cent of the positive cases fall into this age period. The incidence in each age group for the present study, however, is much lower than for the previous investigation.

Table 2 also shows that the number of examinations made in both studies in each age group is comparable for each group except for the groups which include people over twenty years of age. This latter group in the present series is not comparable and does not constitute a representative sampling of the population over twenty years of age.

A study of the relationship between the age incidence and intensity of infestation is shown in *Table 3*.

From this table it can be seen that the same relative proportion of light and heavy infestations for the entire series of examinations is found in each age group throughout the school ages. There is no significant increase in the intensity of infestation as the incidence in the various age groups increases. It has been stated that the age groups above twenty in our series are not representative. The abnormally high proportion of heavy cases can be explained by the fact that they were selected from family

Table 3

INCIDENCE AND INTENSITY OF HOOKWORM BY AGE GROUPS

	Incidence			Intensity (eggs per 0.01 c. c. feces) Per Cent of Positive Cases in Each Intensity Group			
Age Groups	No. Exam.	No. Pos.	% Pos.	1-6 Eggs	7-25 Eggs	26-125 Eggs	126 and Over
0- 2	714	8	1.1	37.5	37.5	12.5	12.5
3- 4	1118	41	3.6	38.0	41.4	18.0	0.5
5- 6	1917	147	7.6	46.4	38.1	15.0	1.5
7- 9	4469	456	10.2	42.1	37.0	17.5	3.3
10-12	3867	504	13.0	36.7	38.1	19.5	6.0
13-14	1723	270	15.6	34.8	40.0	18.8	5.8
15-19	1122	164	14.6	41.4	33.3	21.3	4.1
20-29	201	18	8.9	38.8	16.6	44.4	0.0
30-39	184	10	0.6	30.0	30.0	40.0	0.0
40-49	114	10	0.7	10.0	40.0	20.0	30.0
50-59	53	3	0.5	66.6	33.3	0.0	0.0
60 and over	37	0	0.0	0.0	0.0	0.0	0.0

groups in which a large proportion had heavy infestations. If a sufficient sampling had been obtained from the older age groups, the abnormally high percentage of heavy infestations would probably not have been found.

ASCARIS

Ascaris has been found in fifty-one of the fifty-two counties studied. The positive cases number 6,636, making an incidence of 26.3 per cent for this study. The incidence of Ascaris found by the Rockefeller Sanitary Commission was 21.3 per cent. This probably is lower than the true incidence, as the emphasis was placed on hookworm during that survey, and it may be that not all of the cases of Ascaris found at that time were recorded. These comparative figures serve to show, however, that the incidence of Ascaris has not decreased. While Ascaris has been found in 98 per cent of the counties studied to date, there is a marked variation in incidence in different parts of the State. The results for East, Middle and West Tennessee are given in *Table 4*.

From this table it can be seen that the highest incidence of Ascaris is found in the mountainous and adjacent hilly sections of Tennessee,

Table 4

DISTRIBUTION OF ASCARIS

Division of State	Total Number of Examinations	Per Cent Positive
East Tennessee	12,508	33.6
Middle Tennessee	7,475	30.1
West Tennessee	5,119	3.4

97.3 per cent of the positive cases being found in these areas. The geographic distribution of Ascaris in the United States was pointed out by Otto, Cort and Spindler (1928) as being confined in the main to mountainous and hilly areas. The distribution of Ascaris in Tennessee for the present study agrees with the general geographic distribution as the three divisions of the State differ in their geography. West Tennessee contains both lowlands and hills, Middle Tennessee is mostly hills which become higher as one approaches the eastern part of the State, and East Tennessee is mostly mountainous.

Fig. 2 shows a comparison of the incidence and distribution of Ascaris found by the Rockefeller Sanitary Commission in 1910-1914 with that found during the present study.

In general, the intensity of Ascaris infestation in the present investigation is highest in those counties with the highest incidence.

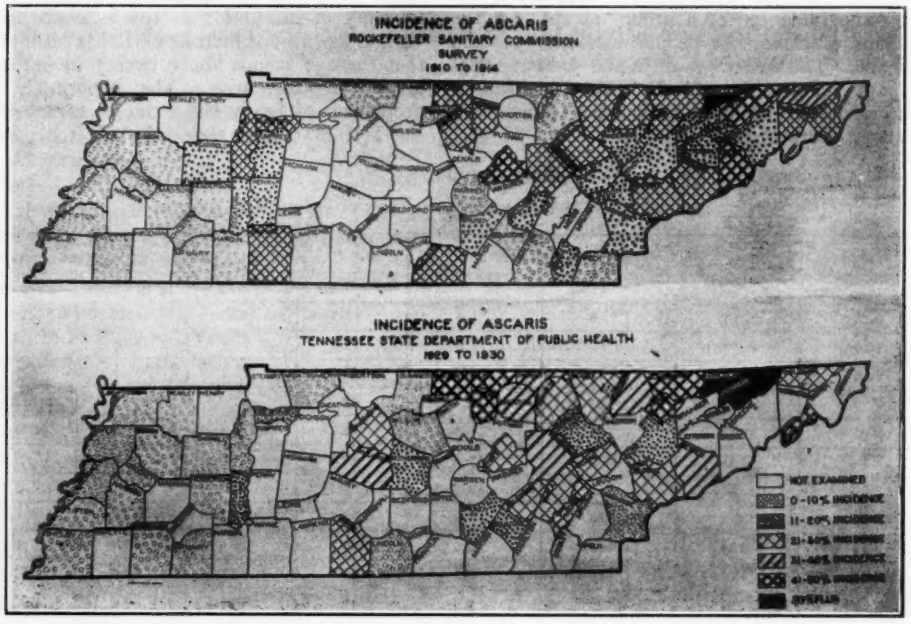


Fig. 2

Ascaris in Virginia was shown by Otto, Cort and Spindler to be confined mainly to the younger children. Our survey shows the same age distribution and it should be pointed out at this time that Ascaris is much more prevalent than hookworm in infants and preschool children.

No control measures have been attempted in Tennessee against Ascaris except along the lines of general rural sanitation. It is interesting to contrast the change in incidence of hookworm since 1914 with that of Ascaris during the same period. The incidence of hookworm has decreased from 25.4 to 7.4 per cent, whereas the incidence of Ascaris has perhaps increased from 21.2 to 26.5 per cent. Even if all of the cases of Ascaris found in 1914 were not recorded, there has probably been no decrease in the incidence of this helminth. It would seem that the factors which are responsible for the decrease in the amount of hookworm either as a result of natural causes or by reason of control measures are not effective in the eradication of Ascaris. These findings would also suggest that in planning control measures against Ascaris, certain biological differences of the two parasites should be taken into consideration. These differences are:

(1) The hookworm egg requires a special type of soil for development, while the Ascaris egg will develop in any type of soil.

(2) The Ascaris egg is more resistant to external environmental influence than the hookworm egg. It has been definitely shown that Ascaris eggs may remain viable in the soil for more than a year.

(3) The Ascaris worm produces 200,000 eggs per day, while a *Necator americanus* produces only about 9,000 eggs per day. Worm for worm, therefore, pollution of the soil with eggs is about twenty times as great for Ascaris as for hookworm.

(4) The usual portal of entry of Ascaris into the body is the mouth, while the most common portal of entry of hookworm is the skin.

Therefore, it is necessary to take into consideration the above facts in attempts to control Ascaris infestation. The use of pit privies by all members of the family is of primary importance. In constructing privies in rural areas, special accommodation for young children should be provided. Careful personal hygiene and education of every member of the family, together with the prevention of soil pollution of the immediate environment, are as important as the construction of privies and should be emphasized.

TRICHURIS

The incidence of Trichuris in fifty-nine counties in Tennessee investigated by the Rockefeller Sanitary Commission in 1910-1914 was 4.7 per cent. The incidence found during the present study is 7.8 per cent, which indicates that there has probably been no decrease in the number of cases of Trichuris infestation. The distribution of Trichuris over the State is practically the same as that of Ascaris, but its incidence is much lower. *Table 5* shows the relative incidence of the two worms in the three great divisions of the State.

Table 5

COMPARISON OF THE INCIDENCE OF TRICHURIS AND ASCARIS

Division of State	Number of Examinations	% Positive for Ascaris	% Positive for Trichuris
East Tennessee	12,508	33.6	9.9
Middle Tennessee	7,475	30.1	9.4
West Tennessee	5,119	3.3	0.35

Fig. 3 shows a comparison of the distribution and incidence of Ascaris and Trichuris in Tennessee at the present time.

This difference in incidence may be explained on the basis of a smaller egg production for Trichuris than for Ascaris. It has been estimated that a female Trichuris will produce approximately 6,000 eggs per day. This factor in itself would indicate that there is less chance for infestation than in the case of Ascaris.

It has been shown by Spindler, furthermore, that the Trichuris egg is not so resistant to external environmental hazards as the Ascaris egg, the moisture requirement of the soil being much greater in the case of Trichuris. This factor may also help to explain the relatively low incidence of Trichuris as compared with Ascaris even though both parasites are taken into the body through the mouth. It is not uncommon to find families with heavy Ascaris infestation, but with no Trichuris. In most instances where such findings are present, the moisture in the soil around the houses is much less than in families harboring both parasites. Excessive shade or poor drainage is found more frequently where both Ascaris and Trichuris are present.

HYMENOLEPIS NANA

The incidence of *Hymenolepis nana* found in the Rockefeller survey in 1910-1914 in Tennessee was 2.4 per cent. During the present investigation 750 cases have been found, making the

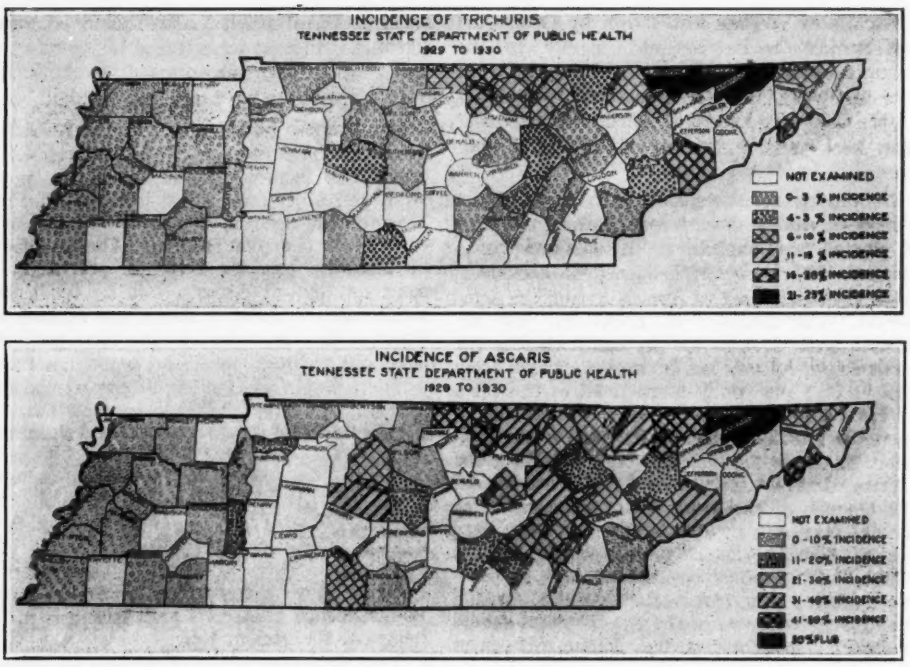


Fig. 3

incidence at the present time 2.9 per cent. Since the egg of *Hymenolepis nana* is infective at the time it leaves the host and probably has but slight resistance to external influences (Spindler 1929), infection is dependent mainly on hand-to-mouth transmission. Our findings indicate that the conditions responsible for transmission have not materially changed during the past fifteen years, if the findings of the Rockefeller Sanitary Commission are correct for this parasite. The geographic distribution of this parasite is somewhat more uniform over the State than that of Trichuris, as can be seen by *Table 6*. Still it is three times as prevalent in East Tennessee as it is in West Tennessee.

Table 6

INCIDENCE AND GEOGRAPHICAL DISTRIBUTION OF TRICHURIS AND HYMENOLEPSIS NANA IN TENNESSEE

Division of State	Number of Examinations Made	Per Cent Positive Trichuris	Per Cent Positive Hymenolepsis nana
East Tennessee	12,508	9.9	3.8
Middle Tennessee	7,475	9.4	2.7
West Tennessee	5,199	0.35	1.2

OTHER PARASITES

During this study, specimens containing eggs of the pin worm, *Enterobius vermicularis*, have been found in only 62 instances, giving an incidence of 0.25 per cent for this parasite. This is a very low incidence for this helminth, especially among population groups which have a relatively high incidence of other parasites which are acquired as a result of hand-to-mouth infection. Incidence figures for this parasite are not available from other parts of the United States, but it is stated (Chandler) that it is one of the most common helminths of man. The failure to find its eggs more frequently is undoubtedly because its habitat is usually the lower colon and the eggs, therefore, are not uniformly distributed in the stool specimens.

Seven cases of *Hymenolepis diminuta* have been found, all in Middle and East Tennessee. This finding is interesting because the parasite is exceedingly rare in man, only about seventy-five cases from all over the world having been reported up to the present time. A separate report on this parasite will be made at a later date.

A diagnosis of Taenia infestation has also been made in three instances.

SUMMARY

(1) A study of fecal specimens from 25,192 individuals has been made among rural white and negro population groups in fifty-two counties of Tennessee. There has been a marked decrease in the incidence of hookworm infestation during the past fifteen years, while the incidence of Ascaris, Trichuris and *Hymenolepis nana* have remained the same. These findings would indicate that the biological differences between these helminths and the conditions of transmission constitute important factors in their dissemination in the general population.

(2) Hookworm has been found in forty-three of the fifty-two counties. The incidence of hookworm infestation at the present time is 7.4 per cent for the series of examinations. The incidence found by the Rockefeller Sanitary Commission in 1910-1914 was 25.4 per cent.

(3) Seventy-nine and five-tenths per cent of all of the cases of hookworm are localized in eleven counties which are situated on or contiguous to the Cumberland Plateau, which is one of the three sandy soil provinces of the State.

(4) Intensity studies show that 77.2 per cent of the cases have from one to twenty-five eggs per 0.01 c. c. of feces and 22.7 per cent have more than twenty-six eggs per 0.01 c. c. of feces, showing that light infestations are found three and one-half times as frequently as heavy ones.

(5) Ninety-four and four-tenths per cent of all the positive cases of hookworm fall into the school age group (5-19).

(6) The incidence of Ascaris is 26.3 per cent for the present series, while the incidence in 1914 was 21.3 per cent. Ascaris has been found in fifty-one of the fifty-two counties studied.

(7) Ascaris is confined mainly to Middle and East Tennessee, 97.3 per cent of the cases being found in these areas which are the hilly and mountainous sections of the State.

(8) The incidence of Trichuris at the present time is 7.8 per cent. The incidence found by the Rockefeller Sanitary Commission was 4.7 per cent.

(9) The geographical distribution of Trichuris is the same as that of Ascaris.

(10) The incidence of *Hymenolepis nana* at the present time is 2.9 per cent. The incidence found by the Rockefeller Sanitary Commission was 2.4 per cent.

(11) The incidence of *Enterobius vermicularis* as found in this study is lower than the actual incidence in the general population.

(12) Seven cases of *Hymenolepis diminuta* were discovered in the present survey.

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DISCUSSION (*Abstract*)

Dr. Paul Eaton, State Board of Health, Jacksonville, Fla.—In Florida we examined 70,000 specimens of feces in three years and 25 per cent were positive for hookworm. We are in a worse plight than Tennessee.

We have found in a county located near Hillsborough County in the year 1927 that nearly 800 of our fecal specimens were positive for Ascaris; in 1929, less than 200. What has happened, I do not know. Nobody has been able to point out why Ascaris has diminished in that one county.

Dr. Joseph W. Mountin, Washington, D. C.—The first point I wish to make is the necessity for defining all public health problems as Dr. Keller has the hookworm problem of Tennessee. All too frequently health officers attempt to apply some public health measure without specific knowledge of the situation or of the probable effectiveness of the projected program. If the expected result is attained it is attributed to the remedial measure. If, on the other hand, the end is not accomplished the procedure is unjustly condemned.

In certain parts of the South hookworm may be even a greater public health problem than it is in Tennessee. I hope that I may be pardoned in going beyond the scope of this paper to stress the importance of control measures which shall embrace both sanitation and intensive treatment. Sanitation must precede treatment if the program is to be effective. Such a plan is now being carried out in Tennessee and intensive work is be-

ing done in those areas which have been pointed out by Dr. Keller.

Dr. Keller (closing).—I have nothing more to add except again to call to your attention the marked reduction in the incidence of hookworm during the past fifteen years. The hookworm problem at the present time involves only about 7 per cent of the population which has been studied. On the other hand, there has been no decrease in the incidence of Ascaris during this period and in all probability there has been some increase in the prevalence of this helminth. Ascaris at the present time in Tennessee probably represents a more important problem than does hookworm, and in developing control measures the biological differences and the epidemiology of the two parasites should be taken into consideration.

VESICAL DIVERTICULUM*

By JOSEPH H. SMITH, M.D.,
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This anomaly occurs often enough in urological practice to merit our attention to its diagnosis, etiology, symptomatology and treatment. The condition is undoubtedly quite frequently overlooked by those not adept in the use of urological diagnostic methods. Its location, size and orifice is at times so concealed that even in the hands of experienced cystoscopists direct observation is often difficult, and it is easily overlooked in the cursory cystoscopic examination. In this paper I wish to recall to your minds some of the salient points as to diagnosis, and the divergence of opinion as to the etiology.

It is generally conceded that vesical diverticulum occurs as a result of over-distension of the inflamed bladder on account of obstruction to the outlet (prostatic obstruction or stricture) and consists in the bulging of the wall of the viscus in the inter-trabecular spaces or any portion thereof.[1]

Diverticula are cavities closely connected with the bladder and resulting from an expansion of a portion of its walls. They are characterized by: (1) the presence of a clearly defined opening which forms a communication between the cavity of the bladder and that of the diverticulum; (2) absence of ureteral opening; and (3) a mucous lining which is continuous with that of the bladder and that of the diverticulum and covers the whole inner surface of the pouch.[2]

Cabot is of the opinion that the word diverticulum should be confined to those cases of pouches, always congenital in origin, occurring most frequently in certain positions but occasionally seen in almost any portion of the bladder and not due to defective development or lack or closure of any recognized structure. He later states that the acquired type must obviously be due to obstruction in the urinary tract at some point below the bladder; and that it behooves the advocates of the theory that they are acquired to produce evidence of obstruction, as it behooves those who hold to the view that they are congenital to show that these patients are not guilty of having an obstruction. He seems further to hold that they are far more probably of congenital origin.[3]

Keyes states that most diverticula are congenital. He argues that the typical large diverticulum usually shows symptoms before the age of prostatism and often occurs in a patient who has no history of venereal disease or evidence of stricture.[4] Judd and Scholl believe that diverticula are primarily embryological defects, while Hinman seems to believe that they are all acquired.

The opinion of the foreign school of urology, as represented by Cesare Boretti (Milan) in his article on "Diverticole della Vesica Urinaria (Pathologia e Clinica)" is as follows:

> In 1923 or 1924 the French Association of Urology and the Italian Society of Urology made a study of the question of diverticula of the bladder and the reports with their discussions presented a complete resume upon this important question. The points brought out dealt more with the pathology of this condition than the surgical treatment. The work of M. Boretti dealt with the study of 45 cases in the clinic of Professor Lasio. The article takes up the etiology and admits as incontestible the fact that congenital diverticula are more or less in the minority. The author comes to the conclusion that there is a congenital predisposition of the vesical muscles. He studies three factors which work toward the formation of diverticula: the anatomical, mechanical, and pathological factors. The increase in vesical pressure is the possible cause of the formation of the diverticulum anatomically. Congenital and acquired forms could not be differentiated. After discussion as to treatment, he reports the results obtained in the clinic of Professor Lasio. Of 37 cases operated upon out of 45 examined, there were 87 per cent cures.[4]

So we see that there is still quite a divergence of opinion among authorities as to whether or not diverticulum is congenital in origin or acquired. Eisendrath and Kolnich say that the middle ground has the most advocates at the present time. Quoting them:

> "It is to be assumed that there are weak spots in the muscular wall of the bladder of certain individuals which permit of herniation of the epithelial and other coats, when obstruction at the vesical neck or urethra occurs. That obstruction plays an important part is evident from the fact that diverticula are rarely

*Read in Section on Urology, Southern Medical Association, Twenty-Fourth Annual Meeting, Louisville, Kentucky, November 11-14, 1930.

found when such an obstruction is absent. Even in young adults and children contractures of the vesical neck have been found by Beer and Hyman to occur more often than was formerly suspected."[5]

Occurrence and Situation.—Diverticula appear more often in the male than in the female in about the proportion of fifteen to one, as far as I can check from the statistics. To those who lean toward the acquired origin of diverticula, this should be some comfort. It appears that since the incidence of obstruction in the lower urinary tract is more frequent in the male, the above figures would tend to bear out their contention. Stricture, whether of congenital or acquired origin, and prostatism, no doubt, are responsible for a certain percentage of occurrences.

The most frequent site of appearance is not far behind and lateral to the ureteral orifice, although it may occur in any portion of the bladder wall. The sac often contains the emptying ureter and that side made inaccessible to catheterization. Due to adhesion of the sac around the ureter and consequent obstruction, hydro- and pyo-nephroses are not uncommon sequellae. Often the orifice to a diverticulum is very small and easily overlooked. At times, under extreme pressure, the opening can be seen gradually to dilate. I do not believe that diverticula are diagnosed from physical findings or physical examination or the clinical symptoms, except in the cases of extremely large ones where a tumor mass is palpable. In fact, those that I have seen, I confess that I was more or less surprised to discover them. Generally they are discovered when the urologist is making a thorough examination seeking the cause of pyuria or cystitis. Cystoscopy should not be relied upon entirely, but regular cystograms should be made which sometimes will disclose a diverticulum otherwise easily overlooked.

Hyman reports in reviewing 600 cases, 20 to 30 in children. Hinman found in 163 cases, 135 single diverticula and 28 multiple. Lower and Higgins state that diverticula occur most often proximal to the right ureter. In their series there were 108 cases in men and two in women. Judd and Scholl, with 133 cases, report 131 in men and 2 in women; Toulson, among 15 cases, finds 1 woman; Durrieus, with 118 men, reports 9 women; Cassuto reports 90 per cent in men, 10 per cent in women, the acquired type being most prevalent.

Calculi.—Judd and Scholl with 133 cases, report stone in the diverticulum in 6 cases; stone in the diverticulum and bladder in 9 cases; Hinman, with 190 cases, reports stone in the diverticulum in 5 cases.

Malignancy.—Judd and Scholl, with 133 cases, report a malignant condition in 10 cases. Lower and Higgins report 1 malignant carcinoma.

Rupture can occur from infection, inflammation and back pressure. Legeau reports such a case.

Symptoms and Diagnosis.—Difficult urination or the sensation of incomplete emptying of the bladder, frequency, burning, occasionally hematuria, feeling of fullness, difficulty in starting urination, passing of foul smelling, thick urine, and occasionally retention, are the principle symptoms. I believe the symptom of not completely emptying the bladder is the most frequent. Urethritis is the symptom that often first prompts the patient to consult the urologist. Upon checking, what the patient had taken for a probable gonococcic infection, the true cause is found to be some obstruction with diverticulum. Absence of gonococci in the microscopic examination in these patients always immediately draws our attention to the upper urinary tract.

The diagnosis can be proven by (1) cystography, (2) cystoscopy, (3) contrast cystograms, (4) coiling catheter in the diverticulum, (5) at times tumor mass can be palpated.

Treatment.—The treatment is largely operative, either by intra or extra-vesical removal, or removal of the lower urinary tract obstruction, such as stricture or prostatic enlargement. These patients are as a rule opposed to the dissection of the diverticulum and in a number only palliative measures can be instituted. In some cases where the retention and size of the diverticulum causes only a minimum amount of distress, relieving the obstruction will often give surprising relief.

REPORT OF CASE

S. G., a white man, thirty years old, radio operator, complained of urethral discharge and painful urination for the previous two years, especially just after urination, with tenesmus of the bladder. He had a spell two or three times each month. Drinking plenty of water seemed partially to relieve his symptoms. There was irritation at the neck of the bladder. The urine passed in a normal stream. There was slight incontinence when the bladder was overdistended. Straining before urination started severe pain. There was no hematuria. Pain at times radiated back to the right flank. The urine was occasionally very foul, with an ammoniacal odor. He had a discharge from the meatus. He gave a history of having been treated for gonococcal infection several years before, also a history of indefinite kidney trouble for the previous fifteen years. Lately he had suffered from constipation and indigestion. He had influenza three years previously.

Physical examination showed a well built healthy looking man. Physical findings were normal. Blood pressure was 130/60. There was no tenderness over the abdomen.

Laboratory Findings.—The total phthalein was 95 per cent in two hours. The Wassermann was negative. Urinalysis showed occasional epithelial cells, occasional

small clumps of pus cells, a few motile bacilli, and 3-5 red blood cells to a high power field.

Cystoscopy and Cystography.—There was vesical neck obstruction, and cystitis. The left ureter was catheterized easily. On the right, what I took to be the right ureteral orifice was catheterized. After the cystoscope was withdrawn cystograms were taken and the catheter found coiled on the right side. Cystogram showed a second diverticulum on the left posterior side and above the left ureteral orifice, simulating a fold of the bladder wall.

Cysto-ureterogram showed little constriction in the left ureter, with two diverticula in the bladder. One almost posterior and the other on the right side lateral to the opening of the ureter. The posterior one was apparently about 3x3 cm. The smaller one was 1.5x1.5 cm. in size. Operation refused.

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Discussion follows paper of Dr. Ockerblad, page 349.

VESICO-INTESTINAL FISTULA: THREE CASE REPORTS*

By NELSE F. OCKERBLAD, M.D., F.A.C.S.,
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There has been nearly 500 cases of vesico-intestinal fistula reported in the medical literature of the world in the past fifty years. This is not so much an index of the prevalence, but rather of the interest manifested in this strange and rare malady. The symptoms are so clear-cut and so absolutely diagnostic that, from the history alone, a second year medical student ought to be able to figure out the correct diagnosis. Perhaps because of this simplicity the disease is frequently overlooked and incorrectly diagnosed. The presence of fecal material in the bladder and mixed with the urine, the passing of gas through the urethra, and the appearance of urine in the stools are the three symptoms that make the diagnosis of this disease certain. All three of these symptoms may not be present at the same time or in the same patient, but the finding of fecal material in the urine and the passing of gas by urethra are most constant and trustworthy symptoms.

The cystoscope is valuable in locating the fistulous opening on the bladder side and the x-ray is a great aid in finding the portion of the intestine that is adherent to the bladder, but neither cystoscope nor x-ray is necessary to establish the diagnosis. The two together confirm the diagnosis.

There have been many excellent reviews on the subject in recent years. The term "vesico-intestinal fistula" was used instead of the term "entero-vesical fistula" by Parham and Hume in 1909, who stated that they considered it a better term than the hybrid Greek and Latin "entero-vesical fistula." The term thus used includes all those pathological communications between the bladder and urethra and any part of the intestinal tract from the anus to the pylorus. Numerous cases have been reported of communication between the appendix and bladder. A few cases of fistula between the small intestine and the bladder have been recorded.

Not the least interesting of these was one reported in 1926 by Knapper and Barthelink, in which a gauze sponge had been left behind in the course of a pelvic operation for uterine tumor. The sponge created a communication between the small intestine and the bladder. A large calculus formed about the vesical end of the sponge. When the sponge was removed at operation a year later and the bladder and small intestine were closed the patient died of peritonitis.

The etiology of this distressing lesion has been well covered in the reviews of Parham and Hume, Bailey, Craig and Lee-Brown and others. A composite of their thought of the causes is about as follows, giving the order of frequency:

(1) Diverticulitis of sigmoid
(2) Malignant neoplasms
  Bowel
  Bladder
  Uterus
  Prostate
  Ovary
(3) Appendiceal abscess
  Pelvic abscess (gyn.)
  Pressure necrosis of peritoneal drainage tube
(4) Traumatic
  Childbirth
  Fractured pelvis
  Operative accidents (litholopaxy)
  Penetrating wounds of rectum
  Gunshot wounds
(5) Prostatic abscess
(6) Dysentery, probably amebic
(7) Tuberculosis
  Actinomycosis
  Syphilis
  Bilharziasis

Chute pointed out that in these cases there may be symptoms in the pre-perforative stage

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Fig. 1
An example of the pre-perforative stage of vesico-intestinal fistula. A bladder diverticulum is shown adherent to an intestinal diverticulum. There is as yet no communication between the bladder and gut.

which are difficult to diagnose. The bowel may become adherent to the bladder without there being an actual fistula. Such cases give rise to bladder symptoms that are difficult indeed properly to account for. I have seen three cases in which bladder symptoms predominated in carcinoma of the large intestine, because of the adherence of the neoplasm to the bladder, although there was no fistula.

Diverticulitis of the sigmoid or colon is by all odds the most common cause of vesico-intestinal fistula. Without going into detail it may be said that diverticula of the sigmoid are frequently found at autopsy and probably many cases of acute diverticulitis have been passed over undiagnosed or incorrectly diagnosed. Only rarely does a diverticulum of the sigmoid adhere to the bladder, and in the course of time and inflammatory storm, finally form a fistulous tract. Malignant disease of the large intestine at any point from sigmoid to cecum is a not uncommon occurrence, but it is rather unusual for such a growth to be placed in a position favorable to the formation of a fistulous tract. Malignant disease of the bladder only on very rare occasions produces vesico-intestinal fistula. Carcinoma of the uterus, the prostate or ovary constitutes a very limited number of the total vesico-intestinal fistulae, although the possibility is there and the mechanism easily understood. As to the appendiceal abscess, I am convinced that there are many more cases in which the appendix adheres to the bladder and causes symptoms whether or not a fistula is formed than have been reported, for I have heard of more cases by word of mouth than I have found in the records.

Pelvic abscesses are a rare cause, but a not uncommon result, of vesico-intestinal fistula. Of the three cases I am reporting, one came for relief of stone in the bladder and because I went into the history rather carefully, I discovered that the patient had a vesico-sigmoidal fistula. The second case was a woman of sixty, in whom the disease had existed for more than ten years, although it had never been suspected that she had a vesico-intestinal fistula. The third case was one of advanced carcinoma of the sigmoid which had not been correctly diagnosed, although it had been present for many months.

Case 1.—The patient was a white man, aged 64 years. He was a small, wiry type of individual 5 feet 4 inches tall and weighing about 118 pounds, who came primarily for the relief of a cystitis. He was brought to an x-ray man by his physician and the x-ray plate showed a large stone in the bladder. A cystoscopy was done following this and because of the number of cellules on the floor of the bladder, we thought at once of a diverticulum. At this time the only history we

Fig. 2
The fistulous opening is depicted by means of a cystogram and as shown above the 5 per cent sodium iodide solution has passed through the fistula and outlines the sigmoid and colon back to the ileocecal valve.

had was what had been told to the radiologist by the country doctor, which was essentially as follows:

About one year previously this patient had what his physician thought was a prostatic abscess and, although he had fever and was quite ill, he kept on his feet and kept going. The physician massaged his prostate with the rather astonishing result that the patient passed a quart of pus by urethra with consequent relief of pain and fever. After that the prostate was massaged several times. He got along well until about six weeks before my examination, when he began to pass blood in his urine, and, although he had had frequency and burning and pain in his bladder region for a year, the appearance of blood in his urine worried him a great deal and brought him back to his physician again. Upon the completion of the cystoscopic examination and the cystograms, the following additional history was obtained. The patient had had trouble with his bowels for more than ten years. He had had attacks of cystitis during that period frequently. For almost a year he had been carrying a bottle in his pocket to collect what he very aptly called "seepage," namely, a continual dribbling from his urethra. He also stated upon interrogation that he often passed gas by his urethra, but he attached very little importance to that phenomenon. He frequently passed urine by rectum, but that did not make much of an impression on him. However, the passing of a substance that looked like fecal material by urethra did cause him a lot of worry. He stated that he has passed fecal material by urethra for the previous two years at varying intervals, but more often in the previous few weeks.

Cystoscopy showed a large phosphatic stone in the bladder. The opening to the fistula was at the bottom of one of the larger of the rather numerous cellules that were scattered about the base of the bladder. The fistula was in the midline about 5 cm. above the trigone. The cystogram shows the bladder outline and the opaque medium has leaked out through the fistulous opening into the sigmoid and outlined the sigmoid and colon to the ileocecal valve. Under sacral anesthesia, a litholopaxy was done, thus clearing the bladder of the stone. We did not care to operate upon this patient in view of the advanced age, the duration of the disease and his appearance of an evidently poor surgical risk. He was much relieved by having the stone removed, for his cystitis cleared up and his dribbling or what he termed "seepage" ceased.

He returned in two months and upon cystoscopy I found two objects that proved to be fecal masses coated with urinary salts, having the appearance of calculi. These were removed by means of rongeur forceps. His hemoglobin was 84 per cent, red blood cells 4,400,000, white blood cells 6,900, and the urine was loaded with pus. On several occasions it became necessary to remove these encrusted fecal masses from the bladder by means of the rongeur forceps. The patient kept begging for an operation.

With some misgivings this was undertaken. With the patient anesthetized and in the Trendelenberg position, a midline incision was made and the inflammatory mass, which included the bladder and sigmoid, identified. A surprisingly large surface area was included in the adhesion between sigmoid and bladder. After packing off the intestines with tape sponges, the bladder was separated from the sigmoid. The opening in the sigmoid appeared to be small, but was surrounded by an area of thickened inflammatory tissue which greatly increased the thickness of the gut wall.

This opening was closed by stitching around the sigmoid so as not to constrict the lumen of the gut. Several layers of chromic sutures were placed. The bladder opening was closed by turning in three layers of fine chromic sutures. A stab wound was made in the left flank for drainage. Three large cigarette drains were placed down in the area of the closure and the abdominal wound was closed. An extraperitoneal opening was made in the bladder and the drainage tube was secured by a purse string suture. The patient went through the operation in good condition and continued in a satisfactory condition for ten days. At a time when we had the feeling that all was well he began to have distention and, since this could not be reduced by any means at our command, we performed a cecostomy under local anesthesia. He gradually failed and died on the sixteenth day.

The pathologist reported that transitional epithelium of the bladder type lined the fistulous tract from the bladder into the sigmoid, the tract being about 5 cm. in length.

At no time did the temperature go above 101° F., and most of the time it varied between 99 and 100°.

On the eleventh postoperative day blood chemistry findings were as follows: non-protein nitrogen 56.4, sugar 175, chlorides 450.

The autopsy showed a large pelvic abscess and peritonitis, which was the cause of death.

Case 2.—A white woman, aged 60, widowed, was seen in consultation to determine the cause of persistent pus in the urine.

She had had a fistulous opening in her left lower quadrant for the previous ten years, from which there sometimes exuded a small quantity of fecal material, gas and urine. She had numerous small superficial abscesses all over her body, which had been present over ten years. There had been many upsets or what her physician had described as "pyemic showers," which seemed to be the starting point for an exacerbation of the multiple superficial abscesses. Two years previously she had an exploratory laparotomy, but apparently nothing was accomplished and nothing found. Cystoscopy showed a double ureter on the right and a single ureteral opening on the left. There were two fistulous openings in the bladder, one about 3 cm. above the left ureteral orifice and another in line 3 cm. above the first. These were about 1.5 cm. to the left of the midline and appeared as raised inflammatory areas with a necrotic center. On the surface of the abdomen on a line drawn from the umbilicus to the left anterior-superior spine of the ilium there were seen two small slit-like fistulous openings which, as described above, discharged fecal material, pus, urine and gas on occasions. The urine was loaded with pus and clumps of pus. Hemoglobin was 70 per cent, red blood cells 3,620,000, white blood cells 8,000, and the urine showed 277 pus cells per cu. mm. The left kidney showed 27 pus cells per cu. mm. The right showed 18 pus cells per cu. mm. Blood culture showed no growth in three days. The Wassermann was negative. This patient was no subject for surgery, and in view of the fact that she had probably gotten along for at least twelve years with the condition, we decided to leave her alone surgically. She lived about a year and died of a brain abscess. Autopsy and section of the mate-

rial which composed the inflammatory mass showed it to be tuberculosis.

Case 3.—A white married woman, aged 44 years, was seen in consultation because she persisted in the idea that she passed gas by her urethra. Her relatives did not believe her and her physician told her it was hysteria. However, the cystoscope revealed the fistulous opening on the right lateral wall of the bladder about 5 cm. from the trigone. A cystogram was made, but none of the opaque media passed through the fistula. After the diagnosis was made the patient gave the following history.

She had passed gas by her bladder for the preceding three weeks and her bowel movements had not "been as they used to be" for more than eighteen months. During that time she had been having two to six bowel movements daily. The stools showed mucus and fresh blood at times. She had bled from her rectum several times during the preceding year and during that time the stools had been black in color. She had had a large amount of flatus during the previous year. She had had urinary frequency and especially nocturia two to three times for the preceding five weeks. Twelve years previously she had the right tube and ovary and appendix removed.

Laboratory Findings.—The urine showed albumin, 1,-600 pus cells per cu. mm. and a few red blood cells. Blood count showed 4,400,000 red blood cells, 10,600 white cells and 90 per cent hemoglobin. The Wassermann and Kahn were negative. Blood chemistry findings were: sugar 122 mg., non-protein nitrogen 35, and chlorides 440 mg. per 100 c. c.

At operation an annular carcinoma of the sigmoid was found which perforated the bladder. This was excised and an end-to-end anastomosis was done. The patient died on the sixth postoperative day of peritonitis. The autopsy showed that the anastomosis had been torn apart by large fecal masses, causing a pelvic abscess and generalized peritonitis. There were secondary matastases to the liver.

There is much to be learned from a study of these three cases, representing as they do three different etiological factors, namely: diverticulum, tuberculosis and malignant disease.

The prognosis in cases of vesico-intestinal fistula is apparently dependent upon the nature and location of the intraperitoneal cause of the malady. The outcome is in no small degree dependent upon the mode of surgical attack. It is generally admitted that the surgery of the sigmoid is the most difficult of all gut surgery.

There are certain principles which stand out in dealing with vesico-intestinal fistula. It seems to me to be of primary importance to divert the intestinal drainage away from the field of operation by inserting a drainage tube higher up in the gut. Another step of importance is to pack off the opening in the gut which has just been sutured, using iodoform gauze and making use of the omentum to form a fistulous tract to the surface in case the sutures do not hold. A third principle, I would say, is to divert the urine by suprapubic tubes in the male and by retention catheters in the female in order to relieve the bladder of pressure.

Albano stated in 1926 from a study of 432 cases collected from the literature, that 1.79 per cent of cases healed spontaneously, that 71.42 per cent of cases healed under medical treatment, and 72.91 per cent were closed after surgical treatment. Twenty-five per cent of the cases are in women.

Only two modes of treatment are possible in these cases, namely: medical or surgical.

The so-called medical treatment consists for the most part in arranging for a low residue diet, bladder irrigations and colonic flushings. The surgical treatment may be palliative, making use of such measures as cecostomy or colostomy, or a serious endeavor may be made to cure the disease by closing the fistula. We must weigh the danger of operation against the dangers and discomfort of the situation as we find it.

Certainly the earlier these cases are seen the better the hope for a surgical cure.

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DISCUSSION (*Abstract*)

Papers of Dr. Smith and Dr. Ockerblad.

Dr. Robert E. Cumming, Detroit, Mich.—I remember while I was in the Army three cases of this sort: two of them were gunshot cases in which the bladder and the bowel had been injured and where such fistulae had resulted. In the third case, both the bladder and the urethra were involved. In this latter case, I implanted the ureter in the bladder and thought I had succeeded, only to have to do a nephrectomy, because of ascending pyonephrosis. In another case in the Army, the condition was admitted to be carcinoma of the rectum, but because of bladder symptoms the patient was referred to us. He was relieved, at least temporarily, by colostomy, which I think is the most useful measure whether the treatment be palliative or curative.

I have seen such fistulae in two cases after hysterectomy, one after a vaginal and the other after an abdominal hysterectomy; and in two other cases, one

following radium implantation, and the other following resection of the bowel with death due to pyonephrosis.

My most recent case is another patient with carcinoma of the rectum. Following resection, the incision refused to heal, and feces came through the bladder. The right ureter was found injured near the bladder. He had a colostomy of course, and we were able to get excellent healing by double-ligating the ureter on that side. After six months the man was sent for, and when he appeared he was almost moribund with a blood nitrogen of 400 mg. and a high creatinine reading. I suppose he would have died shortly, but by means of a left pyelotomy we have temporarily relieved him. Evidently a recurrence of the growth has blocked the remaining ureter.

Colostomy is a most important step in relief of these cases, even from a urological standpoint.

Dr. Thomas R. Huffines, Asheville, N. C.—I want to report a case of a man sixty years of age, who had passed no urine through the urethra for fifteen years. He had a hypertension of 240 over 130. In Atlanta, some advertising doctors had made a "rectal examination." They had found some hard material protruding against the rectal wall and had made an incision in the rectal mucosa after introducing a speculum into the rectum, and made an attempt to pull this material out through the rectum. The patient states they were not successful and a sinus resulted with practically all of the urine coming through this sinus into the rectum. When I saw him quite a cystitis had developed. X-ray and instrumentation located a large stone in the posterior urethra. Later on, after bed rest and under spinal anesthesia, perineal incision was made and a large stone was removed from the urethra; it had not only been in the urethra, but extended into the rectum. The man gave a history of syphilis thirty-five years before. Antiluetic treatment was given and after a secondary operation similar to one for perirectal abscess, the perineal wound and the hole in the rectum closed. The points of interest in this case were: the patient had gone on living for fifteen years with a stone in the urethra; there was a history of an attempt to pull the stone through the rectum, and from that time on he had never passed urine through the urethra until we removed the obstruction. The urethra now carries a No. 26 sound with little difficulty.

Dr. H. K. Turley, Memphis, Tenn.—In our hands the anesthetic of choice has been spinal, using spinocaine. There is perfect relaxation of muscles with easy exposure of the bladder. The bleeding is less and there is less evidence of shock. The patient is usually able to begin taking fluids upon return to his room. Very rarely is there any nausea.

Tatum, of Brooklyn, has a small light mounted on a No. 7 catheter, which is connected with a dry cell battery. This instrument is passed through the cystoscope and the end with the light passed into the diverticulum. Then the light of the cystoscope is turned off and the one in the diverticulum turned on. This gives some estimate of the size of the diverticulum by transluminination.

Dr. Rex E. Van Duzen, Dallas, Tex.—I would like to report a case which I have had under observation for the last six or eight months, a woman about fifty years of age. Another physician reported that he had found several diverticulum-like defects on the posterior wall of the bladder. However, cystograms made by him failed to show any deformities. When I saw her, about a month later, the symptoms were pain in the left renal area and recurring attacks of cystitis. On cystoscoping her, I found what we would term a stricture of the urethra of about 18 French caliber; she had a large cystocele anterior to Mercier's bar; there were two small cellules posterior to the left ureteral orifice, and three posterior to the right. I was unable to demonstrate any of these to be a diverticulum, either by probing or extreme distention of the bladder. This woman also had what Hunner would term a stricture of the ureter. I cystoscoped her every week or ten days, and then later once a month. I saw at the first examination, also on the posterior wall, two small umbilicated papules, and these seemed to disappear as I distended the bladder. Over a six months period these have increased; the first ones have gone on from papules until they are now definite cellules; others have appeared on the posterior wall, some lateral to either ureteral orifice; and one month ago she came in, and on cystoscopy I found that one cellule posterior to the left ureteral opening was a definite diverticulum. I was unable to pass a catheter into it and the x-ray showed a loop about two inches in diameter. In other words, I watched this case develop from a pseudodiverticulum to a definite diverticulum in about six months.

What are these so-called papules that I saw? I removed what I could with a rongeur. It showed nothing but ordinary epithelium. I feel that they are nothing more than redundant mucosa, and on extreme distention they become the lining of the cellules, or they may become the lining of a herniation between the muscle fibres.

I do not believe that obstruction explains it all, neither do I believe that congenital defects, as Rose points out, explain it. Possibly some of these cases are neurotrophic.

Dr. Ockerblad (closing).—All the surgeon has to do to take the conceit out of him is to tackle a few of these cases; and I am sure that all of you will see some of them in the course of your practice. Every one will probably see one, at least, before he dies; and I hope I have seen all I am going to see.

BRONCHIAL OBSTRUCTION FROM INTRABRONCHIAL GRANULATIONS IN A CHILD TWO YEARS OLD: CASE REPORT*

By JAMES B. STONE, M.D.,
Richmond, Va.

Bronchial obstruction in children due to a foreign body is a common occurrence. Reports of obstruction from other causes in infants and young children, though not rare, have been relatively infrequent. The obstruction may be of intrabronchial or extrabronchial origin.

In the young the most commonly reported extrabronchial cause has been enlarged tracheo-

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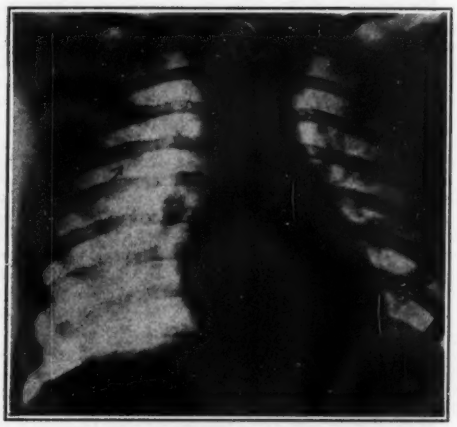

Fig. 1

August 1, 1927—Right-sided emphysema. Barely perceptible small round shadow in right upper lobe obscured by end of third rib.

bronchial and bronchopulmonary lymph nodes. Lerche, in 1827, reported having found in the literature records of 43 cases of ulceration of suppurating tuberculous lymph nodes into the trachea or bronchi. A number of authors have called attention to these enlarged nodes as a factor in the compression and obstruction of the air passages. New growths of the mediastinum or in the lung may similarly cause obstruction. Though these are not common, Rosenson, in 1923, found records of 38 cases of new growths of the mediastinum in infants and children. Tumors of the bronchus have been reported. It has been pointed out by Funk and Clerf that narrowing of the lumen may be brought about by inflammatory conditions of the mucous membrane leading to swelling of the mucosa, collection of thick tenacious secretions, cicatricial contraction or exuberant granulation tissue.

The case here reported falls under the latter head and the clinical picture presented was that of obstruction due to a non-opaque foreign body.

The patient, a white boy, was born June 29, 1925. He first came under my observation in July, 1926, for treatment of an acute gastrointestinal condition.

His family history was negative; his mother and father were both living and well, and so far as known the child had not come in contact with any communicable disease.

He was born at full term, with normal delivery. He had been breast fed exclusively for four months, then given breast and condensed milk until eight months old, after which he had been given a whole milk mixture. His general health had been good up to the time of the above mentioned gastrointestinal attack, which was quite severe and lasted about six weeks.

Subsequent to this he was in excellent health until the middle of July, 1927, at which time he had a mild attack of diarrhea, lasting for several days. The general physical examination at that time did not reveal anything of note.

On August 1, 1927, two weeks after the above mentioned examination, I was called to see the child on account of a slight cough and wheezing, which was noted especially at night. There was no sudden onset, choking nor similar symptoms.

Physical examination showed a well nourished child in good general condition. He did not appear acutely ill. The rectal temperature was 100°.

The tonsils were slightly enlarged.

The chest showed slight signs of rickets, although cod liver oil had been taken regularly during the preceding fall, winter and spring.

Lungs.—Over the right lung anteriorly and posteriorly there was marked diminution of breath sounds. Aeration was estimated as not over 50 per cent of normal. There was distinct hyper-resonance throughout. A few scattered sonorous and sibilant rales were heard.

Examination of the left lung was negative. The rest of the examination was essentially negative.

The signs of unilateral emphysema, developing within such a brief period in a child in good general condition, who had previously shown no evidence of pulmonary trouble, at once suggested the partial occlusion of the primary bronchus by a foreign body.

The blood Wassermann was negative and other laboratory findings were not significant.

X-ray examination was made by Dr. D. D. Talley, Jr. He reported a generalized emphysema of the right lung with a slight limitation of the diaphragmatic excursion on the right side, and considered the findings

Fig. 2

October 17, 1927—Faint shadow extending outward and upward from the hilum to small round shadow at level of end of third rib. Aeration of two sides equal.

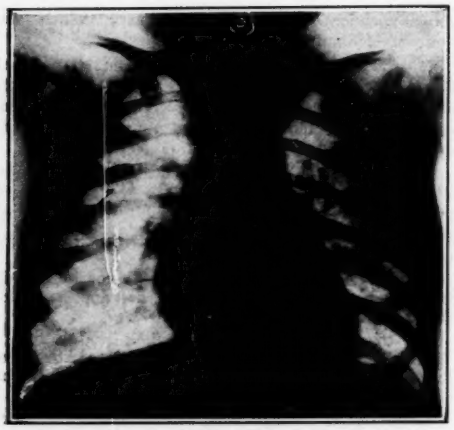

Fig. 3

January 21, 1928—Shadow extending from the hilum out into the upper right lung has cleared. Small round shadow at the level of the end of the third rib is more sharply defined. There is slightly greater aeration of the right lung than of the left.

suggestive of a partial occlusion of the right primary bronchus by a non-opaque foreign body.

The child was then seen in consultation by Dr. Dean B. Cole. He agreed that a bronchoscopic examination was indicated.

Throughout this time there were practically no subjective symptoms. A slight cough and slightly wheezy breathing occurred at times.

The child was sent to the Bronchoscopic Clinic at the Jefferson Hospital in Philadelphia and, on August 8, a bronchoscopic examination was made by Dr. L. H. Clerf. The report of this examination was as follows:

"The mucosa of the trachea was found moderately congested. The orifice of the right main bronchus was found almost completely occluded by an inflammatory mass which was pedunculated, being attached to the outer aspect of the bronchial orifice. A portion of this was removed, after which the bronchoscope was passed into the right bronchus, inspecting the stem bronchus and orifices of the bronchial branches. There was no evidence of foreign body. There was no evidence of bronchial compression. A very small quantity of thin purulent secretion was found."

Bronchoscopic diagnosis: "Stenosis of the right main bronchus produced by a mass of granulation tissue."

The child was kept in the hospital several days, and then returned home. Tuberculin tests, von Pirquet and intradermal, done at this time and repeated about two weeks later were strongly positive.

In subsequent communications Dr. Clerf wrote (September 8, 1927): "Our first thought following bronchoscopy was that there might have been a bronchial foreign body present which produced the pathological findings noted in our report. Such a bronchoscopic picture is not uncommonly observed. There was no foreign body found after a careful search of the tracheobronchial tree and we naturally concluded that a foreign object, if previously present, had been coughed out."

November 7, 1928: "The tissue removed at bronchoscopy proved to be granulation tissue. The case suggests that there is enlargement of the peritracheobronchial lymph nodes which is very probably tuberculous in origin. The granulation tissue was either of inflammatory origin or indicated the point where an infected node discharged its contents into the bronchus."

After returning home, following the bronchoscopic examination, the child began to have fever, fluctuating around 101 and 102°, and had a moderately severe cough. Examination of the chest showed numerous sonorous rales throughout the right lung, though aeration was much improved. The febrile period lasted between two and three weeks. Subsequently, he has been in excellent general health except for a few intercurrent acute respiratory infections usually of the nose and throat, and presumably similar to those generally prevalent during the winter months.

Physical and x-ray examinations indicated progressive improvement in the aeration of the affected lung, though during the six or eight months immediately following bronchoscopic examination there were times when there seemed to be a slight recurrence of the bronchial obstruction, with slight emphysema of the right lung.

X-ray examination by Dr. Talley on October 17, 1927, five weeks after the bronchoscopic examination, showed no evidence of obstructive emphysema at that time. However, a faint shadow was noted in the upper right chest at the level of the third rib in front, extending from the mediastinum outward about half way to the periphery to connect with a rounded, more dense shadow about 1 cm. in diameter lying near the level of the third rib. In the light of this finding a review of the films taken before bronchoscopy showed this small rounded shadow to have been present at that time, though less dense and almost completely masked by the density of the overlying shadow of the third rib.

The faint shadow is believed to represent an inflammatory condition, probably a tuberculous infiltration, existing between the mediastinum and a primary focus of infection indicated by the small round shadow.

The child has had good home care and has been watched closely from the standpoint of diet and hygienic management. He was given ultraviolet light

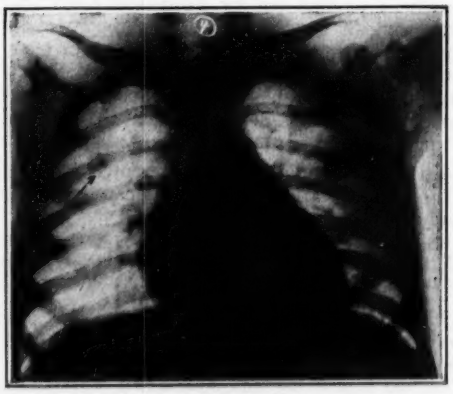

Fig. 4

April 16, 1928—Aeration is equal on the two sides. The small round shadow in the upper right lung is more distinct.

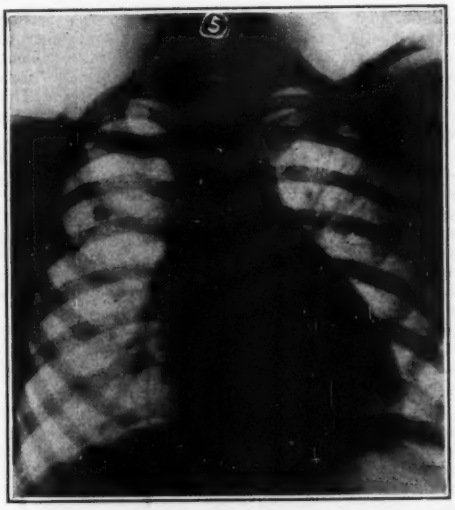

Fig. 5

May 29, 1930—The small round shadow in the upper right lung is completely calcified. There is a slight increase in fibrous tissue formation around the right hilum. Aeration of the two sides is equal.

treatments during the winter following the discovery of his pulmonary trouble, has had cod liver oil regularly except during the hot summer months, and has been given as much outdoor life as the weather conditions permitted.

The last x-ray examination was made on May 29, 1930. This showed some increase in fibrous tissue formation around the right hilum. The small rounded shadow in the upper right lobe was completely calcified and definitely smaller than before. The evidence of infiltration between this shadow and the hilum was no longer present. The aeration of the two lungs was equal. Neither x-ray nor physical examination indicated the presence of any active pathological condition.

He has gained steadily and has developed as a normal child. He has always been very active physically and unusually alert mentally. At the present time, at five and a half years of age, he is in excellent general condition and clinically shows no evidence of any activity of the tuberculous infection.

COMMENT

There is some question as to the significance of the shadows described in the second and subsequent x-ray reports as occurring in the upper chest. The small rounded shadow, now calcified, is believed to have been the primary focus of infection, or it may have been a lymph node that became secondarily involved as the infection extended through the lymphatic system. The area of increased density noted in the second x-ray picture, extending between the mediastinum and the small rounded shadow, probably represented an extension of the tuberculous process from the primary focus to the hilum.

Although the biopsy on the material removed from the bronchus did not identify it as tuberculous, it seems probable that its proliferation resulted from a tuberculous infection of near-by tracheobronchial or bronchopulmonary lymph nodes.

To summarize briefly, this case presents the following points of interest:

A child two years old in apparent good health developed an acute unilateral emphysema. Physical and x-ray findings pointed to obstruction of a primary bronchus by a non-opaque foreign body.

Bronchoscopic examination revealed a pedunculated mass of granulation tissue in the orifice of the right primary bronchus. Following removal of a portion of this mass, there was a brief febrile period followed by progressive improvement and complete clearing of the emphysematous condition.

The child gave a strongly positive tuberculin reaction, and it is believed that the granulation tissue was of tuberculous origin. Serial x-ray examinations showed the active primary focus of infection in the lung, the extension of the inflammatory process to the hilum, subsequent calcification of the primary focus, and clearing of the infiltration between this focus and the hilum.

The child now, three years after the onset of symptoms, is in good condition and shows no evidence of active tuberculosis.

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MELANOMATOSIS: A CASE REPORT*

By J. E. KNIGHTON, M.D.,
Shreveport, La.

The report presented herewith is a case of generalized melanomatosis.

The patient, a colored male approximately thirty years of age, was admitted to the Charity Hospital, Shreveport, Louisiana, in an unconscious condition. The relatives who were present knew little of the details of the illness, but the following information was obtained. The first evidence of trouble had been noted five years previously, and was in the form of a small red area near the inner canthus of the right eye. About two years later a physician was consulted, and an operation, the nature of which is not known, was performed on the eye. Six months before the patient was seen at the hospital a second operation was done. Three weeks before being admitted to the hospital the patient became acutely ill, having an average of eight to ten convulsions a day and being more or less comatose most of the time. This condition persisted until death.

Examination revealed an adult negro male lying in bed in an unconscious state, and having an occasional convulsive seizure which involved the muscles of the entire body. Moderate emaciation was noted.

The lids of the right eye were markedly swollen, and could not be opened, making examination of the eye impossible. The left eye presented no abnormalities of the conjunctive or bulbus, and ophthalmoscopic examination revealed no pathological condition in the fundus.

The ears, nose and mouth were essentially negative.

On the left side of the neck, and in the right supraclavicular region there were groups of tumor masses which ranged in diameter from 1 to 3 inches.

The chest presented a deformity which was secondary to dorsal scoliosis. Examination of the lungs revealed dullness over both bases, and moist rales were scattered throughout both lungs. The heart presented no enlargement, no irregularity, and no murmur. The rate was rapid, and the cardiac sounds were quite distant.

Examination of the abdomen, genitals, and extremities revealed no pathological findings.

The laboratory reported the presence of a moderate anemia of the secondary type. Urinalysis revealed the presence of albumen and a few casts, but no melanin was found. The blood Wassermann test was negative.

The treatment given was entirely symptomatic, and death occurred on the sixth hospital day.

Necropsy was performed, and this revealed numerous interesting findings. For the sake of brevity the report of the pathologist will not be given in detail, but the important facts will be noted.

At the posterior base of the left lung there was a black, spherical mass about four inches in diameter. This was not enveloped by lung tissue, but was semi-pedunculated, and limited by a frail capsule. Section revealed a jet black substance with the consistency of jelly within the mass. There were no visible blood vessels. The right lung presented a firmer mass about one inch in diameter in the upper lobe. This area did not contain as much pigment as the mass in the left lung.

The heart was of about normal size, but the wall of the left ventricle showed relative hypertrophy. The aorta presented early atheromatous changes. The papillary muscles of the right ventricle presented several black nodules from ⅛ to ¼ inch in diameter. These were all firmly attached. Within the cavity of the right auricle there was found a spherical mass, black in color, and about one inch in diameter. This mass was firmly anchored to the auricular wall, and was directly continuous with an irregularly shaped black area which was within the auricular muscle. The black discoloration could be seen externally over the corresponding area of the auricle.

The pancreas was large, firm, and bound down, and in this organ four distinct areas of pathological pigmentation were present. These areas presented the same characteristics as those already described, that is, they were jet black in color and of a gelatinous consistency.

The left suprarenal body was completely replaced by a new growth of the above type. Several retro-peritoneal lymph nodes were found to be involved.

The liver presented only one very small area of pigmentation, and the kidneys apparently escaped entirely. There were, however, evidences of chronic nephritis.

Removal of the skull cap revealed congestion of the cerebral vessels in general. As the frontal lobes of the brain were lifted up it was found that on the right side a black gelatinous mass extended from the optic

Fig. 1
Heart.—Arrow points to tumor, auricular wall folded back.

*Read in Section on Medicine, Southern Medical Association, Twenty-Fourth Annual Meeting, Louisville, Kentucky, November 11-14, 1930.

Fig. 2
Section through pancreas. Dark areas are metastatic nodules.

foramen back to the region of the pituitary body and the optic chiasma. This pigmented material completely invested the optic nerve. The mass described was continuous with a similar growth which was embedded in the lower surface of the right frontal lobe of the brain. Beneath this area of the cerebrum the dura was dark in appearance, and incision of this membrane allowed the scalpel to fall directly into the right orbital cavity. That portion of the cranial floor which forms the roof of the orbit had been entirely destroyed, apparently as a result of pressure necrosis.

The right orbital cavity was found to be completely filled with a black gelatinous tumor mass. No vestige of the bulbus oculi could be differentiated, and it is assumed that this structure had been removed at one of the previous operations.

The diagnosis made by the pathologist was melanoma of the right eye with direct extension to the base of the brain, and with metastatic growths in the lung, heart, pancreas, left suprarenal, and in the lymph nodes of the left posterior cervical, right supra-clavicular and retro-peritoneal groups. This diagnosis was confirmed by microscopical study of the various tissues.

Melanoma is the term now most generally used to describe pigmented malignant tumors. Some years ago it was thought that all such growths were of the sarcomatous type, but later it was definitely shown that some were of carcinomatous origin. In other cases the true type of the growth cannot be ascertained. The case here reported was apparently one of the group of sarcomata.

The melanoma is one of the most malignant growths with which we have to deal, being second only to the chorio-epithelioma. Fortunately both such types of neoplasm are relatively rare. The great danger of the melanoma is due to its rapid and widespread metastasis, and this is dependent upon the ability of this type of tumor cell to enter into and be carried over the body by both the lymphatic and the blood streams.

Regarding such cases there is some controversy as to whether the growth is primary in the eye, or secondary to some focus elsewhere, even though that focus be undiscoverable. Both views are supported by eminent authorities, and it seems logical that both possibilities might obtain.

For the type which is apparently primary in the eye, there is little that can be done. Even when enucleation and complete orbital exenteration have been done as early as possible fatality has resulted from metastasis. It is interesting to note that with primary melanoma of the eye metastasis rarely occurs to the other eye.

Metastatic involvement of the eye by melanoma may follow an original growth in any part of the body. Of particular importance in this connection, however, is the pigmented nevus of the skin. A large number of cases of melanoma-

Fig. 3
Section through base of brain. Extensive involvement of right hemisphere is shown.

tosis apparently arise from such an initial focus, and in most cases there has been antecedent trauma of the nevus, either accidental or by such means as scratching. A point well worth considering should be brought up here, that is, might we not be justified in condemning at first sight the pigmented nevus and removing every such lesion by fulguration or cautery? If this could be done before any irritation had occurred it seems logical that a few cases of melanomatosis might be prevented.

DISCUSSION (*Abstract*)

Dr. J. W. Scott, Lexington, Ky.—Many things contribute to the great interest which attaches itself to any discussion of melanomatosis. Among these are the peculiarity of its histopathology, the fact that some of the tumors are on the connective tissue side and some on the epithelial side, the peculiar behavior of the pigment, and also of the metastases, which is perhaps a corollary of the pigment behavior. Particularly is such discussion of interest when a case occurs in a negro. Some three or four years ago Bauer reported 2 cases in negroes. He said at that time that only 14 cases had been mentioned in the literature as occurring in the negro. Strange to say, of these 14, in 11 the primary growth was on the sole of the foot; his two cases occurring in the nailbed. A few months later Weidman reported a case in a negro in which the primary growth was on the sole. This gives rise to speculation as to the influence of normal pigmentation as an inhibitor of the pigmented nevus. It is certainly striking that of these few cases occurring in the negro some 85 to 90 per cent were on parts which exhibit extremely little pigment. This idea is suggested further by the occurrence of pigmented nevi in white horses. All of these things strongly suggest that normal pigmentation may have something to do with the inhibition of pigmented nevi.

As a further corollary of this peculiar behavior of the pigment is the fact that excessive pigment seems to produce destruction of tissue, so that these black cysts which Dr. Knighton has described in his case, are no doubt due to an excessive occurrence of pigment in the metastases with the production of cysts rather than solid tumor particles. Ewing asks whether much melanoma is missed in the negro, on account of the difficulty of picking up something black on a black skin. It would be remarkable if such disproportion as exists in the reported cases should be due to this fact.

A case which illustrates the variation of pigment production in these cases is reported by McFarland of Philadelphia. He examined a tumor which he said bore all the characteristics of a spindle cell sarcoma of the groin which proved to be a metastasis from a nevus on the foot which had suffered trauma. In this tumor it was only after most painstaking search that a few cells carrying pigment were found. As he says, unless extraordinary care had been used it would have been easy to consider this a primary spindle cell carcinoma of the groin.

The primary occurrence in the orbit is interesting. The weight of testimony in the literature I have examined bears out the idea that it is often primary in the orbit.

Dr. Knighton (closing).—We see many interesting phenomena in connection with the original site of development of this type of malignant growth and the distribution of metastasis.

A case was reported in a recent issue of the *Journal of the American Medical Association* in which both eyes were involved by secondary growths, the primary growth originating in a nevus on the patient's back.

It is interesting to note that both eyes were involved in this case, whereas in cases of general melanomatosis which result from a primary focus in the eye, it is very rarely that the other eye is affected.

In the case which I have just reported, although there was a tumor of considerable size at the base of the brain there was no choked disk or other intraocular disturbance in the eye which was not affected by the melanoma.

Although the right auricle was almost filled by a tumor the only abnormality observed by physical examination during life was a rapid cardiac rate and weakness of the impulse which was in keeping with the general weakness of the patient.

PROBABLE PRIMARY SARCOMA OF THE VERTEBRAE, WITH REPORT OF A CURED CASE*

By H. Theodore Simon, M.D.,†
New Orleans, La.

Malignant metastases of the spine are met with comparative frequency while primary new growths are seldom seen. In fact, in writing on this subject Young[1] states that "primary carcinomas of the spine may be considered a pathological curiosity, while primary sarcoma is sufficiently rare to deserve special consideration." Roberts[2] quotes Coley as finding only 12 cases in a series of 12,000 sarcomas.

Since it has been the good fortune of the writer to treat such a case and since one of the men who will discuss my paper is now treating a similar condition, it may be that primary sarcoma of the vertebrae is more frequent than either literature or hospital records would indicate. Is it possible that this interesting and usually fatal condition is being wrongly diagnosed? Kesmodel[3] writes that detection is difficult and the condition is most often diagnosed as tuberculosis.

Unlike malignant tumors in other regions, there is no age period for sarcoma of the verte-

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†Assistant Professor of Orthopedic Surgery, Post Graduate School of Medicine, Tulane University.

brae. It has been found from childhood to advanced old age. Some of the recorded cases give a history of trauma and it is doubtful whether trauma is a direct exciting factor, whether it stimulates growth of a slower growing tumor, or whether it is a mere coincidence.

Symptoms and Signs.—Pain is probably the first and most important symptom. This pain is both a local tenderness and a neuritis with pain along the course of the nerves emerging at the tumor sight. This root pressure also causes early sensory disturbances which progress to paresthesia and later anesthesia. The growth is usually very rapid, destroying the bony structure of the vertebrae and indurating and invading the surrounding tissue, quickly encroaching on the spinal canal and producing typical signs of spinal cord pressure. The cord pressure causes a muscular weakness and spastic gait, increased reflexes and as the pressure further progresses, a complete paralysis. The induration of the soft tissues is seen early and while the typical kyphosis is not present, still this induration with a possible subluxation of the vertebrae due to destruction of the articulations gives a picture which simulates a tuberculous kyphosis.

Diagnosis.—Diagnosis is made from: First, the above mentioned pain and early pressure symptoms; second, x-ray findings which are fairly pathognomonic, namely, rapid destruction of the vertebrae and a lack of destruction of the intervertebral discs; and, third, the Bence-Jones reaction, if positive, is of some help, while a negative report should be disregarded.

Differential Diagnosis.—Tuberculosis is probably the most confusing factor. However, the differentiation is fairly clear cut. In tuberculosis the local pain and radiating sensory disturbances are not so marked, neither are the spinal cord pressure symptoms unless the tuberculous condition is far advanced with marked deformity present or marked abscess formation. Tuberculosis rapidly destroys the intervertebral discs and much less rapidly the vertebrae. Fixation or traction relieves the pain in tuberculosis, but not in sarcoma. Cord tumors are differentiated by the x-ray and spinal tap. Fractures of the vertebra have definite x-ray differences. Syphilis of the spine shows x-ray evidence together with other tests for this condition. Metastatic malignancy is usually multiple and usually a primary area can be found. Again, the metastasis from an x-ray standpoint is in the center of the body of the vertebra while primary sarcoma appears at the pedicle or first at the edges of the body of the vertebra.

The following case is added to about 30 which have been found by the writer in available medical literature:

A. M., a fourteen-year-old white girl, was first seen in consultation at the J. T. Nix Clinic in New Orleans on December 12, 1925. She was complaining of a muscle weakness and an inability to walk. An x-ray plate taken there showed a destruction of a part of the third cervical vertebra. As the patient needed hospitalization she was admitted to my service in Charity Hospital. Here the following history was obtained:

Complaint.—Paralysis of the legs.

Family History.—The father was killed by bullet wound. The mother died of typhoid fever. Two brothers and one sister were living and well. There was no history of tuberculosis, cancer or nervous diseases.

Past History.—Aside from the usual diseases of childhood she had had no illness. She had not commenced to menstruate.

Present Illness.—The illness began about two months previously when the patient noted a slight pain in her neck which gave the sensation of a "cold in the neck." This pain was intermittent during the day, but when she leaned back or extended her head when standing, the pain was relieved. In two weeks she noted a swelling in the right side of the neck, but the pain was no worse. She visited her family physician. An x-ray was taken and apparently reported as tuberculosis for the physician applied a collar to the neck made of a plaster of Paris bandage. During the first week of wearing the collar the pain was relieved. However, after the second week there was a return of pain, and the patient began to be weak and seemed to have difficulty in the use of the upper and lower extremities, falling easily and requiring to be supported when coming home from school. This weakness became worse and in two weeks (two weeks before admission) she was unable to walk except an occasional step.

She had had no cough nor cold, no expectoration, and no swelling of the extremities. Her appetite was good and digestion good, bowels were regular and there was no trouble in micturition. She urinated three or four times during the day and once or not at all at night. There was no burning nor pain. She slept well.

Physical Examination.—A fairly well developed and well nourished white girl lay in bed apparently paralyzed. The musculature was well developed. There was no bony deformity except that the right forearm was out of true anatomical line because of an old healed fracture.

There was no general adenopathy.

The pupils were equal and reacted to light and accommodated.

The patella reflexes were markedly exaggerated on both sides. There was no ankle clonus, but the tendon reflexes were very active. A slight Babinski was present, especially on the left.

Head.—The skull was negative; the ears, eyes, nose and mouth were negative.

Neck.—On the posterior side of the neck just below the skull was an indurated mass; to the right side a mass was also present, which was larger. There was no tenderness and no fluctuation of the masses.

The thorax was small, thin, flat and symmetrical.

In the lungs the respiratory movements and expansion were equal and symmetrical. There were normal

resonances and vesicular respiration throughout both lungs, and no rales.

Heart.—The apex beat was heard in the fifth interspace and within the nipple line. There was no evidence of enlargement. Both sounds were clear and regular in rate and rhythm. There was no murmur.

The abdomen was small, full and symmetrical. There was a slight rigidity throughout the whole abdomen, but no tenderness. There were no palpable organs nor masses.

Extremities.—The patient was able to move the right upper extremity fairly well, but the left upper extremity rather poorly. There was a spastic paralysis of both lower extremities. She could not walk alone, but when assisted walked with an incoordinated gastrocnemius gait. There were marked changes in temperature, tactile pain and sense of position. There was perversion of vibration sense (burning instead of humming).

Gynecological Consultation.—The hymen was intact. Rectal examination revealed a small uterus. The adnexa were not palpable. The pelvis was negative. Urine was negative to all tests, including Bence-Jones reaction.

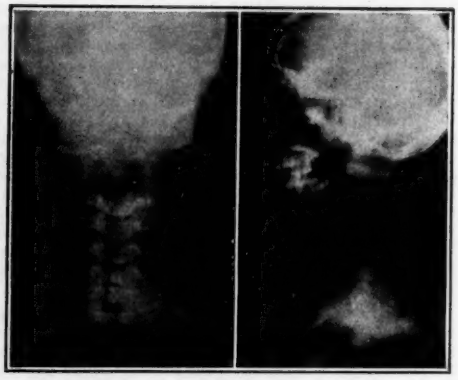

Fig. 1 Fig. 2

A. P. and lateral x-ray of neck showing absorption of pedicle and portion of body of third cervical vertebra. The intervertebral discs are not destroyed.

Fig. 3 Fig. 4

A. P. and lateral x-ray of neck six months after treatment showing calcification of mass and of the destroyed portion of the vertebra.

A spinal tap showed a clear fluid under slight pressure (120 drops per minute). Ten c. c. were removed, which showed a cell count of one, negative globulin and negative Wassermann test. A blood Wassermann was negative.

X-ray plates were reported by Dr. Amadee Granger, of the Radiological Department:

The lateral and right oblique views of the cervical spine show a decided mass on the right and posterior of the third cervical. The lamina and the pedicle of that vertebra are destroyed and apparently replaced by a new growth. The right half of the body of the same vertebra is decalcified. We believe this to be a primary sarcoma of the vertebra.

There was a slight temperature in the afternoon, going to 99.5 to 100°.

Because of the necessity for deep x-ray therapy she was removed to Hotel Dieu January 8, 1926, where additional x-rays confirmed the original diagnosis. By this time the cord pressure symptoms had markedly increased so that she was unable to move a toe and unable to move either upper extremity.

Deep x-ray therapy was begun by Drs. Fortier and Gately, of the Radiological Department. Seven daily treatments were given, a second similar series was repeated in two months and a third series in another eight weeks.

Coley's toxin was given by me, beginning with ¼ minim and gradually increasing to ten minims. The injections were every other day and were accompanied by a slight temperature rise.

Two weeks after treatment was instituted the patient began to regain the use of her muscles, the neck induration gradually disappeared, and very gradually she improved so that when she was discharged on June 8, 1926, she was walking with only a slightly spastic gait and had regained full use of her upper extremities.

X-rays were taken from time to time and showed a calcification of the mass and of the destroyed portions

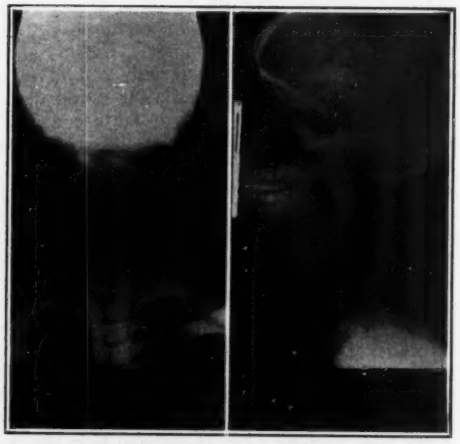

Fig. 5 Fig. 6

A. P. and lateral x-ray of neck four years after treatment showing complete bony ankylosis of the second, third and fourth cervical.

of the vertebra. Plates taken a few weeks ago showed a complete bony fusion of the second, third and fourth cervical vertebrae. The patient at this time is a well developed, nice looking young lady of nineteen with no disability whatsoever. Her walk is normal, reflexes are normal, the neck seems quite normal with a normal range of neck and head motions.

CONCLUSIONS

Primary sarcoma of the spine seems amenable to some degree to deep x-ray therapy and Coley's toxin if this case and similarly reported cases are criteria.

It is possible that some cases diagnosed as tuberculosis may be sarcoma.

Since untreated cases are always fatal, an early correct diagnosis is all-important.

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DISCUSSION (*Abstract*)

Dr. W. B. Carrell, Dallas, Tex.—Dr. Simon's x-rays show a pathological condition in the spine which certainly has the appearance of malignancy. I wish to present three slides of a case with similar appearance in x-ray and gradually increasing absorption of the body of the vertebra and of the processes behind. The first slide is an x-ray of the lumbar vertebra in a woman of forty in whom we diagnosed tuberculosis of the spine. A fusion operation was done. Two months after the operation she developed paralysis, and the x-ray at this time shows increasing absorption of the body of the vertebra and more extensive cyst-like rarefaction posterior to the body and including the processes. In another thirty days a bulging mass had appeared on the posterior surface of the back with slight fluctuation. Needle aspiration showed only fresh hemorrhage, apparently from puncture of the vessels, not extensive. The patient progressively lost weight, paralysis increased, and finally became total from the lesion down. She lived about eight months and died at home.

We made a clinical diagnosis of Ewing's sarcoma and gave her some x-ray treatments, but without benefit. We regretted very much not being able to have a biopsy section or later autopsy, because we are firmly convinced that this case was a sarcoma of the spine, but for lack of proof would hesitate to report it as such.

Dr. Willis Campbell, Memphis, Tenn.—I would like to ask one question: how do you arrive at the conclusion that it is sarcoma?

Dr. Simon.—From an x-ray standpoint and clinical signs and symptoms.

Dr. Jarrel Penn, Memphis, Tenn.—I have never seen a case of primary sarcoma of the spine, but am very pessimistic of the outcome of bone sarcoma in any location. I have tried both deep therapy and Coley's toxin, but all of my cases of true osteogenic sarcoma have resulted fatally. When I see a case of cured bone sarcoma reported I am a little skeptical of the diagnosis.

Dr. Alfred W. Adson, Rochester, Minn.—I wish to drop a suggestion concerning the two plates just shown as primary sarcoma of the vertebrae. In comparing them with plates in similar cases under my own observation I would be inclined to classify them as illustrating foreign-body-giant-cell tumor instead of the malignant sarcoma. Foreign-body-giant-cell tumors respond favorably to either operation or roentgen rays, whereas the truly malignant sarcomas do not respond so favorably.

MALIGNANCY OF THE LOWER LIP, COMPLICATED BY MOUTH INFECTIONS*

By SIDNEY J. WILSON, M.D.,
Fort Worth, Texas

In a search of the voluminous literature on the treatment of cancer of the lower lip, I was unable to find references to such complications as occurred in the cases I wish to report here. For this reason, I thought these conditions might be worth while to discuss as a reminder of some of the potentialities of the septic mouth. The experience related in the first two cases reported is sufficient evidence that the danger of such complications is ever present, regardless of the prophylactic measures taken.

I am indebted to Dr. X. R. Hyde for the privilege of observing the patients in the first two cases reported, and also for his valuable assistance in the preparation of the histories of these cases.

CASE REPORTS

Case 1—L. S., a white man, aged 29, a filling station attendant by occupation, noticed a small nodular lesion on the mucocutaneous surface of the right side of the lower lip in June, 1929. He consulted a physician, who prescribed a stimulating ointment. The lesion grew progressively larger, and in September, 1929, he was referred to Dr. Hyde for treatment. The lesion at that time was hard, infiltrated, and with a granular surface resembling a wart. Clinically, it had the typical appearance of a squamous cell carcinoma. Intensive roentgen ray treatment was instituted and rapid improvement was noted. Four weeks later, the lip was normal in appearance, except for a mild erythema on the mucous surface, which was the final stage of the reaction from irradiation.

Some days later, the patient presented himself with an irritated area on the mucous surface of the lip, which he said had started as a result of a massage in a barber shop. Sodium perborate and improved oral hygiene were advised. The lesion grew rapidly worse,

*Read in Section on Dermatology and Syphilology, Southern Medical Association, Louisville, Kentucky, November 11-14, 1930.

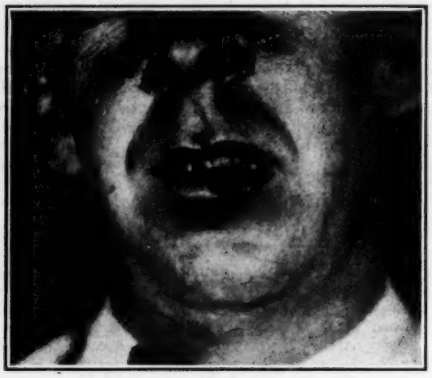

Fig. 1, Case 1
Lesion at beginning of treatment.

Fig. 2-A, Case 2
Lesion at beginning of treatment.

and within one week had spread from the top of the lip to the labial fold, producing sufficient slough to destroy approximately seven-eighths of the thickness of the lip on the mucous surface.

Owing to the appearance of the lesion at that time, it was decided that the original lesion and not a secondary infection was responsible. Fifty milligrams of radium, filtered through dental wax, at 1 cm. distance, was applied for two hours. No changes were noted in the area of infiltration. Local applications of 10 per cent neoarsphenamine, gentian violet, and other antiseptics were continued with very slow progress in the healing of the lesion.

Case 2.—M. M., a white man, aged 54, field manager of an oil company, was seen September 9, 1929, complaining of a small nodular lesion on the left central mucocutaneous surface of the lower lip. He stated that his physician had fulgurated it one month previously. The clinical appearance of the lesion was the same as in *Case 1*. Examination of the mouth showed seven upper front teeth, and one lower left lateral incisor, remaining. The teeth showed marked recession of the gums and alveolar processes, with an exudation of mucopurulent secretion. The other teeth had been extracted on account of pyorrhea.

Intensive roentgen radiation of the lip lesion was instituted, followed by the usual reaction and complete disappearance of the lesion.

February 13, 1930, the lower left lateral incisor was extracted without an anesthetic. This was followed by considerable local reaction and the formation of a hard ring around the socket of the tooth, which the dentist treated with various local applications without effect. In fact, there was some extension of the indurated mass.

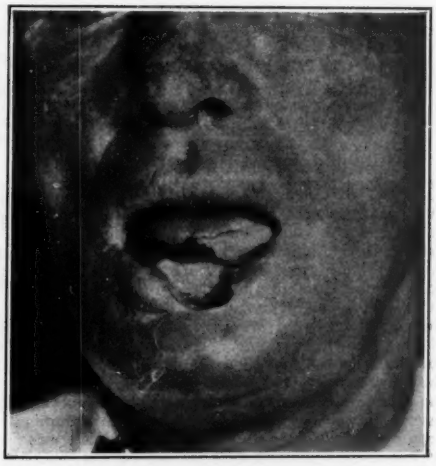

Fig. 2-B, Case 2
Showing destruction wrought by infection.

Fig. 3, Case 3
Lesion at beginning of treatment.

March 19, 1930, 50 mg. of radium was applied to the area of induration around the tooth socket. The reaction was followed by complete resolution of the indurated area, without further treatment.

May 22, 1930, the patient came complaining of severe pain in the lower lip. Examination showed the lip swollen, with painful and deep erythema extending to the chin. Since the patient had been exposed to the sun's rays, it was first thought that this was a secondary reaction from that cause. As the inflammation grew progressively worse, smears were made which showed many pus cells, gram-negative spirilla and fusiform bacilli, with gram-positive diplococci and vibrios. The various local applications used in the treatment of Vincent's angina were applied, without satisfactory results. Within a week's time, more than one-third of the central portion of the lip was destroyed, and the necrosis had exposed 2 cm. of the alveolar process of the lower jaw. The floor of the mouth and two-thirds of the sublingual surface were covered by a yellowish-green membrane. The breath had a characteristic foul odor. Biopsy from the edge of the lesion showed only necrotic tissue. At this time, the patient developed a severe bronchitis and his general condition became critical, his weight having decreased some fifteen pounds. All local applications except a mild alkaline mouth wash were stopped. Neoarsphenamine, in 0.4 gram doses, was given intravenously at four-day intervals.

Immediate improvement in the physical condition was noted. Bronchial irritation and all throat symptoms disappeared within six days. The destructive process was arrested, and daily improvement of the involved area was rapid. July 20, the exposed alveolar process was removed. August 28, the mucous membrane of the mouth was normal; the patient had regained his weight, and the remaining upper teeth were extracted.

Case 3—J. T., a Mexican man, aged 46, came under my observation September 3, 1929, complaining of a lesion on the upper left central portion of the tongue.

Past History.—The duration of the lesion was four months. It began as a small ulcer. The first physician consulted had had a Wassermann test made which was reported negative. The treatment had consisted of local applications, the nature of which the patient did not know, except that a solution of 10 per cent silver nitrate was used over a period of three weeks. He was then given eight doses of neoarsphenamine during the next two months. The progress of the lesion during this time was progressively worse. He was then advised by two surgeons that the lesion was malignant, and that the tongue should be removed at its base.

My examination disclosed an ulcer, irregular in outline, about 2 cm. long, covered with a yellowish-white membrane over an indurated base. There was lymph stasis of the left anterior half of the tongue, which caused some difficulty in speech. The teeth were in bad condition; many were broken and decayed, and one was projecting into the central part of the ulcer. The gums were spongy and hyperemic. His breath had the characteristic foul odor of Vincent's infection. Smears from the mouth and throat showed gram-negative spirilla, fusiform bacilli and vibrios.

Treatment.—The jagged edges of the protruding tooth were removed by a dentist. The use of tobacco, alcohol, highly seasoned foods and condiments was stopped. Hydrogen peroxide and sodium perborate were used exclusively. Improvement was rapid and, after a ten-day period of observation, the patient returned home. In communications from him three and six months later, he stated that the ulcer was well, but that the swelling of the left anterior half of his tongue had not entirely disappeared.

Such cases as I have reported offer no difficulties in the diagnosis of the Vincent's infection. No other known lesion presents such a typical picture and history. However, the baffling feature uppermost in my mind in each instance, was, is the malignancy yet present, or is the condition strictly an infection? The secondary lesion in each instance had a board-like base. This hard base was noticeable in the original malignant lesion.

Since we were mindful of the possibility of easy contamination of the lesion by the organisms found in the oral cavity, it was at first somewhat difficult for us to realize that the actual condition was an infection. It has been said that the Vincent's group of organisms exist as saprophytes in the oral cavity of a large percentage of the normal population. This group, or certain members of the group, become pathogenic, and the cases reported here are fair examples of their action while pathogenic.

The interesting points in common in these cases were:

(1) An acutely inflammatory reaction;

(2) An over-active salivary system;

(3) Headache;

(4) All of the submaxillary group of lymphatics were enlarged;

(5) General debility and loss of weight;

(6) A characteristic fetid odor; and

(7) The rapidity of the destructive process.

Biopsies and smears in each case failed to reveal evidence of malignancy in the secondary lesions. The smears, on the other hand, presented the usual group of organisms classified as Vincent's (angina) infection.

In all cases presenting a neoplasm of the lips or of the oral cavity, saving of the patient's life is of the first importance. Assuming this viewpoint, I feel justified in resorting to mutilation in order to accomplish the desired results. Yet, in cases wherein poor oral hygiene is evident, the results shown here may prevail regardless of what method has been resorted to to obtain the desired result. Oral hygiene has for a long time been considered very important when treating a neoplasm around the alveolar area, on the lips or the face, or involving the gums. Many times I have used irradiation in treating a lesion in the vicinity of gums diseased with pyorrhea, and

knew full well that the inflammatory process in the gums would undergo an acute exacerbation, but little did I realize what might be the consequence. According to experiments, there are a variety of lesions which may be produced by this group of organisms. The field must be made fertile, which can be done in many ways. In these cases, the field was made fertile by radiation assisted by trauma in the presence of poor oral hygiene.

D. T. Smith[1] claims that 80 per cent of the population have this assortment of organisms in the tonsillar crypts and around the teeth. He reports six cases of pulmonary abscess in which smears from the teeth and from the abscess presented almost identical findings.

Mead[2] states that:

"Vincent's angina is caused by fusiform bacilli and spirochetes in symbiosis. Both of these micro-organisms may be found in normal healthy individuals as saprophytes, but by symbiotic action they may become pathogenic in debilitated conditions or from neglect of the mouth."

Hollander[3] found in 24 consecutive patients presenting carcinoma of the oral cavity that all showed evidences of gross oral sepsis.

In the past, where time was not a vital element, it has been my routine that the mouth and teeth should be attended first, and the strictest oral hygiene observed. If the lesion was on the mucocutaneous surface, the teeth and gums were properly protected from the rays of radium or roentgen ray. I was not unmindful of the potentialities of the septic mouth, as a single experience in treating a neoplasm of the gum in a mouth showing gross oral sepsis will cause one to approach a similar condition with extreme caution.

In reviewing these cases, I am sure that some will feel that the radiation was the cause of the destruction of tissue. If the management of the first case is noted carefully, I believe that it can be definitely shown that this was not true. In this case, only enough radiation was applied to destroy the tissues down to the base of the lesion. This was accomplished through only one port of entry. There was the usual expected reaction, disappearance of the legion and healing. The patient complained of the soreness of the teeth, which is commonly encountered. After the reaction was practically complete, the trauma mentioned in the history occurred. The period of time between the initial roentgen radiation and the onset of the acute symptoms does not speak for irradiation as the cause for the sloughing. The base of the lesion in this case was hard and board-like. The cavity measured 1 by 2 cm. This indurated base caused us to doubt the diagnosis of Vincent's infection and, at this time, 50 mg. of radium filtered through dental wax was applied to the lesion, with no appreciable effect. Had the secondary lesion been caused by the first radiation, the second application would have added fuel to the flame. On the contrary, the lesion healed slowly and the end-result was loss of a large portion of the lip. It is interesting to note that the acute symptoms began in the labial fold, well away from the neoplasm and in the vicinity of the peridontal tissues which became acutely inflamed following the first irradiation. From this point, it spread to the lip margin.

In *Case 2*, the initial lesion was much smaller than that in the first case, yet the destruction was much greater. The amount of radiation applied was just sufficient to destroy the lesion. In this case the dose was smaller. Considerable time passed, as is shown by the history, and a tooth was removed after the secondary condition had become acute. The hard ring developed around the tooth socket and persisted, and in the face of treatment by medication, grew in size. This led us to suspect a metastasis from the original malignancy. Radium, well controlled, was applied by the cross fire method, taking care to protect the part of the lip which had been radiated before. The usual reaction ensued, and the ring of hard tissue disappeared, exposing a small portion of the alveolar process which was very irregular.

The case appeared to be progressing nicely when, about one month later, an acute pain developed in the chin. Sloughing followed immediately and rapidly. In this instance, the sloughing as it approached the lip margin, missed the scar marking the site of the original neoplasm. The sloughing began in this case near the tooth socket and spread both ways. In this second case, a second biopsy was considered. The character of the base of the lesion was the same as in *Case 1*. A second biopsy was not done, due to the recognized dangers thereto. In due course of time, the infection spread to the throat and trachea and, perhaps, to the bronchi. In each instance, the general condition of the patient was quickly changed to a debilitated state.

Considering the two cases in combination (*Cases 1 and 2*), I might add that all of the recognized principles of medication recommended for Vincent's infection were carried out. In *Case 3*, the same type of secondary lesion developed without the probable influence of irradiation. I am unable to understand why it re-

sponded so quickly to simple local applications, because the patient had the typical septic mouth, plus decayed teeth.

It is evident that the pathogenic manifestations of Vincent's infection may be produced in many ways, and this condition should be constantly kept in the mind in the management of oral lesions.

In the many cases of oral lesions that we have treated by irradiation, the complications described have occurred only in the two cases reported here.

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DISCUSSION (*Abstract*)

Dr. M. T. Van Studdiford, New Orleans, La.—Only a few months ago we saw a patient at Charity Hospital who followed very closely the history of *Case 1;* but instead of being a Vincent's infection, the condition was lymphatic leukemia in the aleukemic stage of the disease.

Case 3 reminds me of a patient, a small white girl, suffering with noma. There was a through and through punched ulcer about the size of a 5-cent piece in the right cheek and necrosis of the mandible. The patient recovered after extensive surgical removal of teeth and most of the mandible.

Lately I have seen in both the white and negro wards, patients with arsenical dermatitis following neo-arsphenamine who developed Vincent's infection. Formerly we used neo-arsphenamine in glycerine, locally, for Vincent's.

Dr. Harry M. Robinson, Baltimore, Md.—We have lately had a mild epidemic of Vincent's disease in Baltimore with rather curious effects. This led me to examine normal mouths, as well as those with Vincent's disease. In several of the normal mouths repeated smears were practically normal, although in some in whom spirilla were absent a few small fusiform bacilli were seen. I have found it easier to examine the smears, or drop, by the dark field method. In these examinations I have found that in cases with symptoms there were marked motility and a greater number of organisms than in the normal mouths.

In one of our cases the lesion on the tongue looked so much like a chancre that examination was imperative, but on repeated examinations only Vincent's organisms were found. The lesion disappeared rapidly after one intravenous injection of arsphenamine, but recurred with a similar lesion on the eyelid and around the anus. This patient became rapidly worse, developed marked leukemia and died in two weeks' time.

Another patient was a man with a swollen tongue in the center of which was an ulcer. On the margins of the tongue there was marked leukoplakia. I examined this by dark field and found numerous Vincent's organisms. I did not take the blood for complement fixation test until later when, after repeated treatment, the mouth did not markedly improve. The blood was positive, but in view of the resistance of the ulcer to antisyphilitic therapy this was not considered an important factor. A biopsy taken a few weeks later was shown to be squamous carcinoma, but the Vincent's organisms had practically disappeared.

ALLERGY AS A CAUSE OF GASTROINTESTINAL DISORDER*

By W. W. Duke, M.D.,
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Allergy is important in its relationship to the gastrointestinal tract not only because it can produce serious gastrointestinal disorder, but also because it can simulate both functional and organic disease due to other causes. Consequently, unless recognized, it can lead to gross error in diagnosis. It is important also because the gastrointestinal turmoil caused by allergy can actually precipitate organic disease which may require surgery for relief. It is important, finally, because therapeutic measures for the relief of gastrointestinal allergy are sensationally effective if adequately administered.

Allergy can involve almost every active tissue. It is characterized by certain features which make its recognition relatively simple and certain. A physician can diagnose the condition correctly, as a rule, if he is well acquainted with the subject. Allergy can simulate a large variety of diseases, but rarely simulates any of them perfectly. It should be looked for, therefore, whenever the symptoms displayed by a patient do not fit well with known disease patterns.

PATHOGENESIS AND PATHOLOGY

The typical local lesion of allergy is characterized by anemia and edema of the part directly affected, surrounded by an area of erythema. The typical cell found in the lesion is the eosinophil. This is in contrast to the neutrophil cells associated with acute inflammations and the plasma cell associated with chronic inflammatory processes. Typically, the above lesion constitutes a hive. It is associated usually with sensory disturbances such as itching and, in the gastric tissues, sometimes by pain. In addition to this, allergy may be associated with a disorder which occurs as part of a general reaction of allergy. This consists basically of over-activity of one or more or all the branches of the vagus system. It causes

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anomaly in muscle contraction and in secretion. In the case of widespread allergy, shock may result characterized by disappearance of pulse and respiration, cyanosis, and loss of consciousness. Shock may terminate fatally unless promptly and adequately treated. It is interesting to relate that each of the above phases of allergy can be relieved as a rule within a matter of moments through prompt and adequate use of adrenalin.

SYMPTOMS

Allergy may make its appearance at any point in the gastrointestinal tract from the lips to the rectum, giving rise to the signs of local and general reaction as above mentioned. It can cause symptoms which may be mild or severe, acute or chronic, intermittent or continuous. The extreme symptoms which are occasionally observed are often alarming. Nausea, vomiting and diarrhea may equal that caused by any other disease known to medical science. Edema of the membranes may be so extreme as to cause complete obstruction and finally pain may equal that caused by gallstone, kidney stone, or gastric crisis. Allergic disorders characterized by anemia and edema of the membranes, increased tone of non-striated muscle and increased secretion, can easily precipitate inflammatory disease in the gastrointestinal tract or its appendages. I have observed a number of cases of acute appendicitis which commenced with an attack of alimentary allergy. I believe the characteristic symptom of acute appendicitis as mentioned in the common text books, that is, pain in the epigastrium followed after a day or so by pain and tenderness in the region of the appendix, is frequently caused primarily by gastric allergy, which is later followed by a complicating inflammatory disease of the appendix itself.

DIAGNOSIS

It was mentioned above that alimentary allergy can simulate many functional and organic diseases, but that it rarely simulates any of them perfectly and can almost always be distinguished from the other diseases. It can be suspected whenever alimentary symptoms do not fit well with the usual symptomatology of other known diseases, and especially if the disturbance is associated with other easily recognized symptoms of allergy such as allergic coryza; swelling and itching of the pharynx; hoarseness and edema of the larynx; bronchial cough or asthma; or by skin manifestations such as pruritis, urticaria, angioneurotic edema, or eczema. The condition may also be suspected in the absence of these easily recognized symptoms if a well marked family history or past history of allergic disease can be obtained, or if the condition disappears after a therapeutic test with adrenalin. The severest symptoms of allergy should yield to adrenalin unless complicated by some non-allergic change in the affected tissues. Adrenalin must be administered with precision. Unfortunately, we have no way of determining the ideal dose for a given patient or even for the same patient at different times. It is a good rule, therefore, to give about 0.1 c. c. or a little more at five-minute intervals until tremor appears. Tremor usually indicates that the administration has been adequate and that true allergic symptoms should soon subside. If the dose is pushed to the point of causing cardiac arrhythmia or heart pound, a rubber tourniquet should be placed around the arm above the site of inoculation of adrenalin and released at frequent intervals. An excessive adrenalin effect can be stopped in this way (Insley).

TYPES OF REACTION

Symptoms of alimentary reaction can be divided into two distinct groups: first, those which occur as a result of direct contact between the tissues and the offending agent; and second, those which occur as part of a general reaction caused by contact with a substance after absorption. As an illustration of the former type may be mentioned the violent gastrointestinal symptoms which occur as a result of the ingestion of a small amount of honey in honey sensitive cases. Pain, nausea and vomiting occur as a rule within a few minutes of the time of ingestion of the honey and may be shortly followed by symptoms of general reaction. I have observed a case in which the ingestion of wild honey on two occasions caused gastrointestinal symptoms immediately followed by a convulsion, coma, complete disappearance of pulse and respiration and cyanosis. This patient could tolerate other types of honey. The application of honey to the skin in cases of this sort gives rise immediately to a typical hive. This type of case reacts in the same way that pollen sensitive cases react to the application of pollen.

The second type of reaction (alimentary symptoms occurring as part of a general reaction) is observed typically in the case of alimentary symptoms following an insect bite in hypersensitive persons. Here, symptoms such as nausea, vomiting, bloating, diarrhea and collapse may be very severe and may actually, in rare instances, cause dehydration, collapse, and death.

Both the above types of reaction may occur simultaneously. This is illustrated in pork sensitive cases. Here a person may have coryza and headache and gastric disturbance caused by direct contact between the membranes and emanations from the food as it is eaten and later may have urticaria and other general symptoms occurring as part of a general reaction.

CAUSES OF REACTION

The commonest single cause of alimentary allergy is food sensitiveness. Alimentary reaction caused by sensitiveness to drugs, sera, insects and animal parasites (including intestinal worms) is less common. Finally, as will be discussed subsequently, symptoms caused by sensitiveness to the effect of heat and effort and to the effect of cold are also uncommon. Pollen, epidermal substances, dust, smoke and volatile oils which commonly cause nasal and bronchial symptoms are rarely absorbed in quantities sufficient to produce reactions in the alimentary tract.

Reactions to foods are interesting. Symptoms which are inclined to be chronic and continuous are almost always caused by a food which is eaten repeatedly. Attacks which occur at rare intervals are usually caused by foods which are rarely eaten. It is much easier to make a diagnosis in the latter case because the patient, as a rule, can notice a relationship between cause and effect. In the cause of sensitiveness to foods commonly eaten, such as milk, eggs, and pork, the diagnosis is beset with difficulties, especially if the patient is highly sensitive and reacts to traces of the offensive agent. Infants highly sensitive to egg can actually be made ill by the quantity of egg they receive from the lips of a person who has recently eaten egg. Adults highly sensitive to egg can actually obtain enough egg from the eating of hen meat to be made profoundly ill. I have had two individuals highly sensitive to pork who could be made profoundly ill by the trace of lard contained in a small slice of bread; also one individual who was so highly sensitive to cane sugar that an illness could be produced by placing one or two granules of sugar on the tongue. Several honey sensitive cases who were not sensitive to the gross constituents of honey such as sugar, were made ill by the trace of foreign substance contained in one drop of honey. One ragweed sensitive patient who could tolerate commercial honey of five varieties was made ill by a small quantity of wild honey. This was caused probably by its content of ragweed pollen.

DEGREE OF SENSITIVENESS

Whereas a few individuals are highly sensitive, such as those above mentioned, many patients are slightly sensitive and are made ill only by the eating of definite quantities of the offending agent. This is particularly true in children. Many individuals are brought to a state of invalidism through the eating of normal amounts of milk and eggs and can be completely relieved by their partial avoidance.

PHYSICAL ALLERGY

There is a type of reaction similar in every sense to the allergy as above described caused by specific sensitiveness to the action of physical agents. I have grouped these cases together under the term "physical allergy," meaning altered reactivity to the effect of physical agents such as light, heat, cold and scratches and in the case of heat sensitiveness, to the effect of mental and physical effort. So far as the alimentary tract is concerned, the only reactions of importance are those caused by sensitiveness to heat and cold and to the effect of effort. Cases of physical allergy can be divided into two groups: first, those in which reactions occur only at the point of contact between the tissues and the physical agent, and those in which reactions occur not only at the point of contact but in distant structures as well, in fact, frequently in distant structures only.

Reactions to cold which occur only at the point of contact with cold are rare but interesting. The drinking of water between 5 and 15° C. in cases of this sort may cause nausea, vomiting, and abdominal pain. Exposure to a cold wind may cause coryza and asthma and the application of cold water to the skin may cause a typical hive.

Reflex-like reactions, that is, reactions which occur in points distant from the point of exposure, are quite common and rather frequently involve the gastrointestinal tract. It can cause gastrointestinal symptoms which may be more or less violent, symptoms such as nausea, vomiting, diarrhea, and pain. Attacks may be associated with other symptoms of physical allergy such as prostration, tremor, headache, allergic coryza, asthma, pruritis, urticaria, angioneurotic edema, and occasionally, shock.

Heat reactions of this sort may occur as a result of gross exposure to heat in patients who are slightly sensitive, and as a result of almost infinitesimal exposure to heat in patients who are highly sensitive. In the latter case, a small quantity of heat such as is encountered in the

drinking of a cup of coffee with a meal may precipitate an attack, or the physical effort of getting up out of a chair may precipitate an attack, or the mental effort of an interesting business deal, or the watching of an athletic contest, or an emotional disturbance may precipitate an attack. In a fashionable woman who was highly sensitive to heat, a vivacious conversation was likely to be interrupted by acute diarrhea.

The symptoms in patients who are heat or effort sensitive can be reproduced objectively by the application of heat to the skin. In this way, a diagnosis can be proved. The symptoms can be relieved almost immediately by the application of cold to the skin. Interesting to relate, the symptoms can be produced by heating an extremity such as the arm with a hot lamp even though a tourniquet is applied above the point of application so tightly as to stop the circulation of blood. Evidently the symptoms produced in this case are produced through the sense of heat. Interesting to relate, also, is the fact that an attack generated in this way can be promptly stopped by the application of cold to another extremity even though a tourniquet be applied above the site of application of cold. A patient who is inclined to have symptoms of this sort can frequently relieve himself by as simple a procedure as holding a piece of ice in his hands or immersing his hands and forearms in cold water. The relief which can be obtained frequently in this way in severe cases is nothing short of miraculous.

In the case of reflex-like reactions caused by cold sensitiveness, we have a reverse status, that is, symptoms such as abdominal pain, nausea, vomiting and diarrhea often associated with other allergic symptoms such as prostration, tremor, urticaria, pruritis, asthma, allergic coryza, and so forth, caused directly by the effect of cold on the skin or mucous membranes. A cold drink, a cold food, or cold on the skin may have like effect. Symptoms in this case can be relieved by the effect of heat or effort. Surprising to relate, the symptoms can be reproduced not only by the application of cold to the skin, but also by the application of cold to an extremity even though a tourniquet be applied above the site of application in such a way as to obstruct the circulation. Likewise, relief can be obtained when produced in this way by heat applied to an extremity distal to a tourniquet which completely obstructs the circulation.

Violent gastrointestinal symptoms, therefore, can be caused by an agent of such minor importance as a sense of heat or a sense of cold, and can be relieved by the reverse application.

The cases just described are quite different from the so-called contact group of cases referred to previously. In the contact case of reaction, the reaction occurs only at the site of contact with the agent unless the application is so gross as to cause a general reaction. I have observed several cases of this type which showed marked sensitiveness to cold with marked swelling of the lips and esophagus and abdominal pain, nausea and vomiting after the ingestion of a cold drink or a cold food. In this case the effect of cold upon the skin was negative so far as the alimentary tract was concerned.

SPECIFIC DIAGNOSIS OF ALLERGY

Much more can be gained in the diagnosis of gastrointestinal allergy through the agency of family history, past history, physical examination, tests with heat and cold, therapeutic tests with adrenalin and food elimination tests than through the use of the skin tests ordinarily employed in allergic studies. These facts have been particularly emphasized recently by Rowe, who has written extensively and accurately on this subject.

SPECIFIC TREATMENT

Brilliant therapeutic results can be obtained in alimentary allergy through the agency of elimination diets, specific treatment in the case of sensitiveness to common foods (such as milk, eggs or pork) which are difficult to avoid, and through the use of symptomatic remedies, especially adrenalin, ephedrine, atropine, and the salicylates.

In the treatment of physical allergy much can be gained through avoidance of undue exposure to the physical agent which causes reaction and through the agency of increasing tolerance for the offending agent by repeated gradually increasing exposure to the agent primarily responsible for the illness.

SUMMARY AND CONCLUSION

Allergy is important in relation to the gastrointestinal tract in that disturbances caused by allergy can simulate organic and functional disease and through causing edema of the tissues, muscle contraction, and anomalies in secretion, can actually precipitate organic disease.

Allergy in the gastrointestinal tract is more commonly caused by foods than by drugs, sera, insects, and parasites.

A reaction similar in every sense to those caused by foods or other material agents can

be caused by sensitiveness to the effect of heat and effort or to the effect of cold.

Pollen, epidermal substances, dust, smoke, volatile oils, which commonly cause nasal and bronchial symptoms, are rarely absorbed in sufficient quantities to produce reaction in the gastrointestinal tract.

The diagnosis of gastrointestinal allergy can be made with relative certainty.

Treatment of the condition is likely to be brilliantly successful if the case is correctly diagnosed.

DISCUSSION (*Abstract*)

Dr. Julius Friedenwald, Baltimore, Md.—Food allergy should always be considered in the diagnosis of gastrointestinal affections. In a study of 500 cases of mucous colitis reported from our clinic, this condition occurred in about 1 per cent as a definitely proven causative factor. The affection was due in a large proportion of instances to meat foods, such as chicken, veal, pork; to fish, eggs, milk; to certain vegetables as onions, tomatoes, celery, asparagus, turnips and cabbage, and to certain fruits as lemons, oranges, bananas, grapefruit and strawberries. In one instance each it was due to chocolate and cocoa.

There is at times great difficulty in determining a positive relationship to a specific sensitization in mucous colitis, especially as other factors may play a role and as the patient may not be at once relieved by the withdrawal of the specific food, until other predisposing causes are overcome, such as general indiscretions in the diet, persistence of constipation, and so on. In general, according to our experience, it may be stated that the sooner the allergic factor is determined in this affection the better is the prognosis. For it is easily understood how difficult it may be to overcome conditions of this type following the development of secondary pathological changes, even though the part played by the specific factor is withdrawn.

It is, therefore, of great importance to bear in mind the possibility of the presence of this factor in the early diagnosis as well as in the treatment of mucous colitis and kindred affections, as in a certain proportion of instances food allergy undoubtedly plays a definite causative role.

Dr. H. G. Barbour, Louisville, Ky.—Dr. Duke's paper is extremely interesting to physiologists and particularly to me because of the disturbed regulation of body temperature which occurs in these extraordinary conditions. There is no doubt whatever with regard to the skin allergy which Dr. Duke has illustrated beautifully in his exhibit, that the edema which brought on these sudden attacks might be of reflex nature from the skin or from any other part of the body. Analogous to this symptom of sensitiveness to heat or cold is the way in which fluid can be moved in the body by exposure to cold baths or hot environment. Cold baths shift the fluid out of the blood and some of it undoubtedly goes to the skin and subcutaneous tissue. Every one knows that extreme heat drives the fluid out through the skin. Dr. Duke tells us that in case of skin allergy we have a dry skin; we have a suppression of the sweat cells. In this case fluid contributes to the edema which might otherwise be taken up by the sweat glands. I mention this point in order to ask Dr. Duke whether in gastrointestinal allergy there is any evidence of a suppressed intestinal secretion which might account for the different distribution of the fluid.

Dr. L. P. Gay, St. Louis, Mo.—Dr. George Dock, under whom I was fortunate enough to study medicine, was in the habit of saying that an abdomen has as much expression as a face. Recently I have come to the conclusion that an abdomen can easily put on a false face and mask an allergic reaction as an acute or chronic gall-bladder disease, renal colic, acute or chronic appendicitis, mucous colitis, colonic dysfunction, diarrhea, or practically any type of abdominal discomfort. In my work I choose to refer to the condition as the allergic abdomen, as the symptoms are so varied though arising from a common cause. Symptomatology is often misleading. A careful history frequently gives no lead to allergic susceptibility either by concomitant phenomena or by food likes or dislikes. In such an instance, negative findings associated with an almost invariable eosinophilia are of great value. The ordinary allergic abdomen, however, will occasionally give an associated history of urticaria, arthritis, hayfever, or attacks of asthma. Attacks of unexplained diarrhea are also common. The most common abdominal discomfort is the symptom complex which traveled for many years under the name of dyspepsia nervosa. Patients of this type have a lowered gastric acidity curve. They uniformly have low basal metabolic rates and eosinophilia both in the blood and in the mucus obtained from the lower colon. Their symptoms are relieved by the removal of the specific allergens from their diets, and their symptoms are easily reproduced by purposeful feeding of their individual allergens. On a restricted diet it is usual to find other troubles relieved. For example, some of my patients are always constipated when they have urticaria and have normal bowel movements when they are free from skin irritation. In other words, there is no spasm or edema of the intestinal tract while on a proper diet. It is not always possible to get skin reactions from everything to which the individual is sensitive. If complete relief is not obtained, then the use of a diary is very helpful. In this way many offending foods may be restricted that would have remained unsuspected otherwise.

Dr. Duke (closing).—I have not made a particular study of suppressed secretion. Sometimes we have a very violent diarrhea with food allergy, which would indicate that under certain circumstances the secretions are not suppressed.

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MILK IN ANOREXIA

It is one of the contradictions and extremes of civilization that while a large part of the population suffers from hunger, lives perhaps on garbage and roots, while whole Indian or Chinese villages may starve, while the United States suffers from a depression which endangers the food distribution of the nation, another considerable social group visits the physician to complain that its young, in spite of plenty on the table, with the world market to choose from for its food, is undernourished and has to be coaxed to eat.

Many pediatricians in the past three years have attempted to answer the question of why certain carefully brought up children have no appetite. Looking through the current literature one sees frequently such titles as "Treatment of Anorexia," "My Child Won't Eat," "Factors Involved in Combating the Hunger Strike in Children," "Influence of Excessive Milk Feeding on Gastric Motility and Its Relation to Chronic Anorexia," "Analysis of Anorexia," and so on. The subject has been extensively if not conclusively debated.

Milk, which has been the fortress of nutritional experts for the past twenty years, is being attacked from many sides as a possible source of the anorexia. Milk, the former idol of the pediatricians, is now feeling some of the "stings and arrows of outrageous fortune." There are physicians who wish to dethrone the idol; and milk companies, even, exhibiting the spirit of preparedness, have in some instances begun to bottle orange juice for distribution with milk on their wagons.

Rosamond, of Memphis, some years ago presented a paper before the Southern Medical Association on "Evils of Too Much Milk."[1] Lane and Bosshardt[2] showed last year that children in an institution gained weight as rapidly and looked as well as the controls if a quart of fruit juice a day were given with a vegetable dietary instead of a quart of milk.

Experimental anemia has been produced in rats by nourishing them on milk alone. Milk has been shown by Whipple and Robscheit-Robbins[3] in their series of classic experiments on hemoglobin production in the dog to be conspicuously lacking in hemoglobin-producing ingredients; and anemia is of course one of the common complaints of the age, and one of the frequent concomitants of anorexia.

Logically, one may add, from natural experiments, milk is a food for the toothless.

Lucas and Pryor,[4] of San Francisco, have completed an analysis of 110 children complaining of anorexia, upon whom they have compiled a large amount of data. They conclude that anorexia occurred more frequently in children who are taller than the average and slightly underweight; and that these were frequently given by their mothers a high fat diet, with eggs and milk to increase their weight. They advise the elimination of milk from the diet and substitution of a quart of fruit juice per day instead.

In the causation of anorexia, many factors other than milk must be involved. Lack of exercise and outdoor play undoubtedly assume a large role. Shortage of vitamin B in the diet must be considered. Psychic factors are important. Babies seldom show anorexia. It is the child who has begun to talk and express preferences for certain foods who has no appetite. By failing to eat he gets attention which, according to the psychiatrists, he craves. Urging

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3. Whipple, G. H.; and Robscheit-Robbins: Influence of Dairy Products on Hemoglobin Production. Amer. Jour. Physiol., 79:280, 1927.

4. Lucas, W. P.; and Pryor, H. B.: Factors Involved in Combating the Hunger Strike in Children. Amer. Jour. Dis. Child., 41:249, Feb., 1931.

him to eat, quickly becomes a habit with the mother.

Sweets should be most carefully considered as a source of anorexia. The run-about child wants them and usually gets them, and sugar is the best known satiety producer.

Another thing not usually mentioned in discussions of anorexia is the depressing effect upon the appetite of cod liver oil, which is now commonly given to children.

Milk, by universal agreement among nutritional experts, is the most nearly perfect food in animal nutrition. It contains more of the elements essential to life than any other food which has been investigated. While "excessive" milk feeding, whatever that may be, has been protested from several sides, the matter is not settled of maintenance of a good nutritional state in children without it. The literature of milk's merits is still much greater than that of its defects.

The frequent occurrence of anorexia in children under the pediatrician's care is evidence that the present methods of feeding the young, while vastly superior to those of twenty years back, are ready to progress a little further.

CURABILITY OF SYPHILIS

Cancer is so commonly considered incurable that the average physician feels very reluctant to report this diagnosis to a patient. He more often gives the frightful news to a near relative of the patient, to whom he leaves the responsibility of enlightening or deceiving the victim.

Syphilis, likewise, is considered in many quarters incurable; but with syphilis, for social reasons, usually the diagnosis is given to the patient himself. The more educated the carrier, then, the greater his despair, and the more frequent his threats of suicide.

To the controversy as to whether syphilis is ever cured by modern treatment, Dr. Aldred Warthin,[1] internationally known pathologist of the University of Michigan, has contributed most evidence. In this issue of the JOURNAL[1] are summarized the study of twenty years of autopsy material in which microscopic evidence of latent syphilitic infection was sought more carefully, possibly, than it has ever been sought before. Latent syphilis was found in 25.7 per cent of all necropsies of persons over twenty-five years of age in the University Hospital, at Ann Arbor, in the past ten years. It was demonstrated in one-fourth of all patients over twenty-five years of age who came to autopsy in a Northern hospital, where the incidence of venereal disease is according to popular belief less than in warmer climes such as those represented in the Southern Medical's territory. A large per cent of these patients had received intensive treatment, and some were Wassermann negative. In all patients in whom syphilis had been diagnosed in life, more or less active foci of latent syphilis were found in the tissues at autopsy. In other words, careful examination found no completely cured cases, but explained as syphilitic many deaths which were frequently attributed to other causes.

The sex differences in the localization of the spirochetal foci were of clinical interest. Worse and more frequent heart and cerebral lesions were found in men than in women. The testes of every latent syphilitic male showed evidences of the presence and progress of the infection; yet of thousands of ovaries examined, not one showed a single syphilitic lesion. From these observations, syphilis in a woman should be less a contraindication to marriage and procreation than syphilis in a man. The danger of transmission from a woman may be less, and there may be less danger of infection of the offspring.

This series of autopsies, then, offers no encouragement to the physician who wishes to demonstrate that syphilis is curable. The pathological evidence is wholly against him. One needs here, therefore, to call into the argument William James' old thesis on the "Will to Believe." Faith is rational, says James, when it is desirable. The physician must have faith in the curability of syphilis if he is to carry on treatments. A definite belief in recovery is as necessary to the patient as mercury, bismuth, and arsenic, if he is to undergo the long, troublesome, and frequently painful antiluetic therapy. "The best inspirer of hope is the best physician," said Charcot.

Cases of prolonged clinical arrest of the disease, with apparent health and productivity, and families of healthy children, have been observed. If the incidence of syphilis is as great as Warthin reports, this occurrence must be more frequent than has been realized.

Infection with tubercle bacilli is said to be practically universal, and post mortems of clinically healed cases always show foci of tubercle bacilli. It is common to find little or no func-

1. Warthin, Aldred Scott: The Lesions of Latent Syphilis. Sou. Med. Jour., this issue, pages 273-278.

tional disturbance even when anatomical involvement is relatively great. And tuberculosis is not considered an incurable disease. Since spirochetal lesions are to be found in many stages of activity, it is to be hoped that many of them may have the same significance as the healed or partly active tuberculous lesions, though this has not been proven to be true.

The physician should examine his statistics of office patients, questioning whether it is possible that one of every four of the adults has syphilis. He should hold out to his luetics constantly the expectation of cure, meantime exhorting them to the need of prolonged rest and hygienic measures, along with specific treatment, and a life time of careful observation.

HEALING OF FRACTURES WITH FIBRINOGEN

When a broken bone has been put in the best possible mechanical alignment, and fixed there with bandages and splints, the surgeon usually considers that he has done for the patient all that medical science can require. In certain cases he gives special attention to the soft parts. In some fractures, massage and physiotherapy are employed to prevent stiffness and restore function to the injured member. Systemic measures, such as the administration of calcium and vitamin D or cod liver oil, may be recommended in cases of debility to assist the healing process.

The use of local injections of drugs to expedite bone formation has rarely been seriously attempted. Kugelmass and Berg,[1] after a series of studies of ossification in rabbits, have described a local injection which apparently specifically hastened bony union in a few instances.

Microscopically, they say, the process of ossification consists in, first, the production of fibrous tissue, and second, its infiltration with calcium to form solid bone. Hemorrhage at the site of injury releases fibrinogen from the blood, and healing begins with the formation of a framework of fibrous tissue from the liberated fibrinogen. The initial supply of calcium salts for adsorption is obtained by local formation of amino acid, which dissolves at least 20 per cent of the exposed ends of the broken bones. Elements needed subsequently for bone construction are obtained from the blood stream.

Spontaneous healing, therefore, depends on local hemorrhage induced by trauma to the tissues, and formation and hardening of a fibrous network. The greater the local development of fibrous tissue, the speedier the union.

Trypsin, it will be recalled, is a digestive enzyme which has the power of dissolving fibrin. If, shortly after fracture in a rabbit, an alkaline solution of trypsin was injected by the New York workers into the hematoma at the site of injury, fibrous dissolution was produced, and union markedly delayed. Injection of fibrinogen, on the contrary, accelerated bony union, presumably because it furnished building material for the necessary fibrous mesh. The blood calcium was simultaneously elevated by fibrinogen.

Healing may be hastened, the New York writers conclude, by increasing the supply of fibrinogen at the site of fracture. Bone repair is a local rather than a systemic process, and union may be retarded or accelerated by these local therapeutic injections.

The number of rabbits used by Kugelmass and Berg is not sufficient to establish the result, and of course the healing of broken bones is something that may vary considerably from species to species. It is of interest that any simple and purely local measure seems to expedite or delay the process of fracture union. If their hypothesis is true, however, fractures in which there is most bloodshed should unite more rapidly than those with little bloodshed; and this has never been demonstrated to be the case.

1. Kugelmass, I. N.; and Berg, R. N.: Ossification: Callus Formation and Calcification. Amer. Jour. Dis. Child., 41:236, Feb., 1931.

Book Reviews

Cancer of the Lung and Other Intrathoracic Tumors. By Maurice Davidson, M.A., M.D., B.Ch. (Oxon.), F.R.C.P. (Lond.), Physician to the Brompton Hospital for Consumption and Diseases of the Chest, and Dean of the Brompton Hospital Medical School, Chelsea. With a foreword by Arthur J. Hall, M.A., M.D., D.Sc., F.R.C.P., Professor of Medicine in the University of Sheffield. 173 pages with illustrations. New York: William Wood & Co., 1930. Price, cloth, $5.50.

In this book the author presents an intelligible picture of the essential facts of cancer of the lung. The subject is presented in seven chapters, the first four dealing with the statistical, pathological, clinical and radiological aspects of the disease; and the last three with diagnosis, non-malignant intrathoracic tumors, and treatment.

In the chapter on statistical aspects the author gives a brief account of the early development of cancer and compares the incidence of primary lung cancer on the Continent, in the United States, in Canada, and in Eng-

land. He also quotes figures from Brompton Hospital from 1918 to 1929, inclusive.

Under pathological aspects, he gives a review of the literature and a summary of views of leading pathologists on histological classification of primary intrathoracic growths. In this summary it is interesting to note the changes in histological opinion. In this same chapter he discusses the etiology from the viewpoint of age and sex incidence and occupation. One important fact which he brings out is the high incidence of lung cancer noted among workers in the Schneeberg mining district of Saxony, which is described in detail. He also states that carcinoma of the lung among miners in Czecho-Slovakia is considerable. In this district attempts to cure bronchial cancer by radium produced an acute exacerbation of the whole process. He also mentions tuberculosis and influenza as possible etiological factors.

He discusses the clinical aspects under history and mode of onset, symptoms and signs, differential diagnosis, and course and development. He also reports 22 cases with post-mortem examinations.

Under radiological aspects, he points out the absolute need of cooperation between radiologist and clinician. He gives a resume of the best literature dealing with lung cancer; discusses x-ray findings with reference to clinical and post mortem findings and gives a differential diagnosis from the radiological standpoint. He also mentions the importance of correct centering of the patient and the necessity of duplicating the films.

In the chapter on diagnosis, he mentions all modern methods of examination such as complete history, physical examination of the chest, x-ray, artificial pneumothorax, lipiodal injection, barium meal in case of esophageal obstruction by growth, bronchoscopy, and even thoracotomy.

In discussing non-malignant intrathoracic growths, he gives a short review of the literature and examples of cases with clinical and radiological reports.

Under treatment he suggests possible lines on which treatment may develop in the future, discusses the possibility and limitations of surgery, radium and x-ray, and the palliative treatment of advanced cases.

The illustrations are excellent and the book is worthy of high commendation.

Intestinal Tuberculosis. By Lawrason Brown, M.D., Consultant to the Trudeau Sanatorium, Saranac Lake, New York, and Homer L. Sampson, Roentgenographer to the Trudeau Sanatorium, Saranac Lake, New York. 376 pages with 122 engravings and 2 colored plates. Philadelphia: Lea & Febiger, 1930. Cloth, $4.75.

The medical world has always received with eager anticipation any work emanating from Trudeau Sanatorium. In this, the second edition of their book on intestinal tuberculosis, the authors have followed the general outline used in their first edition, but have thoroughly revised and amplified the material used in the first edition. Of special interest is the chapter on roentgenologic diagnosis, in which they have included the newer work. The plates, too, are exceptionally plain and descriptive.

Traumatotherapy. By John J. Moorhead, B.Sc., M.D., F.A.C.S., (D.S.M.) Professor of Surgery and Director, Department Traumatic Surgery, New York Post-Graduate Medical School and Hospital. 574 pages with 625 illustrations. Philadelphia: W. B. Saunders Company, 1931. Cloth, $7.00.

The author states that the "causation, symptoms and diagnosis of most traumatopathies is apparent from the history, or becomes so on inspection or by the aid of x-rays or other routine laboratory aids." He thus limits his book to the technic of treatment.

The chapters on contusions and wounds follow the basic principles of today. Anti-gas gangrene serum is recommended, but at the same time "it has not proven specific." Anti-tetanic serum is not given intraspinally, intracerebrally or intraneurally, Moorhead feeling that this procedure is valueless. Treatment in most cases is given by the method that the writer has found most efficient, though at times more than one method is detailed. Iodine remains the prime favorite as an antiseptic, either U. S. P., or in a saline solution. Structure in hand injury is discussed in detail; discussion of the foot is not so extensive or complete. Joint injuries are also included in this chapter.

In opening the chapter on dislocations this admonition is given: "The vast majority of recent joint injuries associated with deformity and disability are fractures and not dislocations." Starting with the jaw and carefully going over each part of the skeletal system, the treatment of the different dislocations is described. The treatment is based on the author's four R's: (1) recognition or diagnosis, (2) reduction or setting, (3) retention or immobilization, and (4) restoration or functioning. These are also applicable to fractures.

The chapter on fractures is the most extensive in the text. The apparatus used has been simplified wherever possible, in most cases the molded plaster splint being the supporting agent of choice (sugar-tongs). In compound fractures a pyocyaneus infection is likened to the laudable pus of our ancestors. In fractures of the extremities and also in dislocations, an electrically driven machine called the "repositor" is used in their reduction. An unusual number of methods are given for the maintenance of position, especially for the extremities. The procedure of laminectomy is repeated in this chapter.

Trauma to closed cavities, blood vessels, tendons, nerves and special agents are each given a short chapter. Amputations and plastics, both facial and bone, standardized procedures for first aid treatment and special apparatus are all mentioned. Compensation and the legal complications of accidents are often a bugaboo to the practitioner. The author tells how to avoid these annoyances. He bases his estimate of the amount of damage to a member, grading the restoration of function 60 per cent, union 20 per cent and contour 20 per cent.

The increasing mechanization of the world, especially with the advent of the automobile, and now with the aeroplane has opened the way for the traumatic surgeon. At the same time it has not exempted numberless general practitioners. The abundance of illustrations, excellent descriptions and completeness of this volume make the work most valuable.

The Chest in Children. By E. Gordon Stoloff, M.D., Assistant in Pediatrics (Assistant Radiologist) Mt. Sinai Hospital. Volume XII of Annals of Roentgenology, Edited by James T. Case, M.D., Professor of Roentgenology, Northwestern University Medical College, Chicago. 432 pages. 406 roentgen-ray studies and 19 clinical illustrations. New York: Paul B. Hoeber, Inc., 1930. Price, cloth, $15.00.

This is the twelfth volume in a series of Monographic Atlases entitled "Annals of Roentgenology." It is presented in six sections.

In Section 1, the author discusses the trachea and bronchi under the following headings: trachael stenosis; normal bronchi; bronchitis; bronchial stenosis; and bronchiectasis. He emphasizes the necessity of knowing the normal structures and the mechanism by which obstruction may be caused.

Section 2 is a presentation of the lungs, both normal and pathological. In this he discusses circulatory disturbances; disturbances of aeration; lobar pneumonia; bronchopneumonia; cysts and tumors of the lungs; pulmonary infiltrations of uncommon occurrences; suppurative and necrotic disease of the lungs; and pulmonary tuberculosis. The completeness of this section can be realized by the fact that the subject matter is presented in three aspects, clinical, radiological, and pathological. One hundred eighty-six pages and 197 illustrations are devoted to this phase of the book.

Diseases of the pleura are given under the following classifications: acute pleurisy; chronic disease of the pleurae; tuberculous disease of the pleurae; and pneumothorax, hydrothorax, and hemothorax. This section in its completeness and thoroughness sustains the high standard of the book.

Under section 4, the mediastinum, the author discusses anatomy and physiology; emphysema of the mediastinum; the thyroid; cysts and ganglioneuromata; the thymus; and the intrathoracic lymph nodes. In reading this chapter, one could not supply additional information because every conceivable pathological condition of the mediastinum has been adequately covered.

In section 5, the diaphragm, the author describes the normal diaphragm, and the causes and significance of its elevation and depression.

The final section is given to the chest wall under the following sub-headings: peculiarities of the chest wall; deformities of the chest; congenital abnormalities; and calcification in the chest wall.

The illustrations are excellent in their choice and reproduction and an extensive bibliography is added. The fact that the author is clinically as well as radiologically trained makes this book worthy of the highest commendation and destined to many future editions.

Stalkers of Pestilence. By Wade W. Oliver, M.D., Professor of Bacteriology, Long Island College Hospital; Visiting Professor, University of the Philippines (Auspices of the Rockefeller Foundation). Introduction by Theobald Smith, M.D., Ph.D., Director, Department of Animal Pathology, The Rockefeller Institute. 251 pages, 23 illustrations. New York: Paul B. Hoeber, Inc., 1930. Cloth, $3.00.

This seems rather a brief history of medicine than of bacteriology. It is written in popular style, with large, clear print.

The Treatment of Diseases of Children. With Special Formulas and Drugs for Childhood and a Short Diagnostic Summary of Each Clinical Picture. By Prof. Dr. F. Lust, Director of the Children's Hospital, Karlsruhe. Authorized translation of the sixth German edition with additions by Sandor A. Levinsohn, M.D., Associate Pediatrician to the Barnert Hospital, Paterson, N. J. Attending physician to the Daughters of Miriam Orphan Asylum, Clifton, N. J. 513 pages without illustrations. Philadelphia: J. B. Lippincott Company, 1930. Price, cloth, $8.00.

This book has the inherent defects of most translations from a foreign language, although the author has attempted to adapt it to American needs by copious use of foot notes. One is impressed with the fact that the original work from which the translation was made needed revision in several respects to make it conform to recent advances in pediatrics. The translator has pointed out these defects in his foot notes, which are necessarily very brief. The chapter devoted to therapeutic technic is especially valuable and gives, in detail, the indications for and methods of using all the physical means of treatment.

Part two of the book is devoted to drugs and formulae used in pediatric practice. The drugs are arranged in alphabetical order, and one is immediately struck with the inclusion of a large number of German proprietary drugs which are seldom used in this country. In spite of this, however, this part of the work is complete and gives the indications, dosage and methods of use of all drugs necessary in the treatment of the diseases of children.

Clinical Atlas of Blood Diseases. By A. Piney, M.D., M.R.C.P., Research Pathologist, Cancer Hospital, London; Consulting Pathologist, Chelmsford Hospital, and Stanley Wyard, M.D., M.R.C.P., Physician, Bolingbroke Hospital, and Assistant Physician, Cancer Hospital, London. 98 pages with 36 illustrations, 32 in color. Philadelphia: P. Blakiston's Son & Co., 1930. Cloth, $4.00.

This book contains beautiful pictures showing the morphology of the blood in most of the known diseases rare and common. Excellent illustrations likewise show the derivation of various cells. The volume is convenient in size and make-up.

Ante-Natal Care. Including the Abnormalities Associated with Pregnancy and a Section on Post-Natal Care. By W. F. T. Haultain, O.B.E., M.C., B.A., M.B. (Camb.), F.R.C.S.E., M.R.C.P.E., M.C.O.G., Senior Assistant Obstetric Physician, Edinburgh Royal Maternity and Simpson Memorial Hospital, and E. Chalmers Fahmy, with a foreword by Professor R. W. Johnstone, Professor of Midwifery and Diseases of Women, University of Edinburgh. Second edition. 127 pages. New York: William Wood & Co., 1931. Cloth, $2.25.

The topics of interest reviewed are the diagnosis of pregnancy and its early signs, hygiene of pregnancy, early care and methods of avoiding complications, pelvic measurements and treatment of contracted pelves, pain and hemorrhage, venereal disease, eclampsia, albuminuria and nephritis.

The Pathology of Internal Diseases. By William Boyd, M.C., M.R.C.P., Ed., Dipl. Psych., F.R.S.C., Professor of Pathology in the University of Manitoba; Pathologist to the Winnipeg General Hospital, Winnipeg, Canada. 888 pages with 298 engraved illustrations. Philadelphia: Lea & Febiger, 1931. Cloth, $10.00.

The purpose of this book is to present the gross microscopical and functional pathology of internal diseases, and correlate the symptoms with the pathological changes. A synopsis of its contents will show the scope of the book: diseases of the heart, arteries, respiratory system (larynx, bronchi, lungs and pleurae), stomach and duodenum, intestines, liver and gall-bladder, pancreas, kidneys, adrenal glands, thyroid gland, parathyroid gland, pituitary body, the blood, bone marrow, spleen, lymph nodes, thymus gland and nervous system. The book is modern and thorough. Because it was written as a companion to the author's "Surgical Pathology," certain sections as "Diseases of the Gall-Bladder and Stomach" are not given in the same detail as other parts of the text. The author follows a definite system in presenting his subject which enhances the value of the book. A bibliography is added to each section. The illustrations are well chosen and executed. The text is clearly written and the author's command of English makes the book pleasant as well as instructive reading.

Modern Methods of Treatment. By Logan Clendening, M.D., Professor of Clinical Medicine, Lecturer on Therapeutics, Medical Department of the University of Kansas, with chapters on special subjects by H. C. Anderson, M.D.; J. B. Cowherd, M.D.; H. P. Kuhn, M.D.; Carl O. Rickter, M.G.; F. C. Neff, M.D.; E. H. Skinner, M.D.; and E. R. DeWeese, M.D. Fourth edition. 819 pages with illustrations. St. Louis: C. V. Mosby Company, 1931. Cloth, $10.00.

In this revised edition the sections on myxedema, diabetes, coma, diet in tuberculosis, cisterna puncture, and infantile paralysis have been rewritten. The chapters on hydrotherapy, diet, psychotherapy, medical gymnastics and exercise make the book more valuable. Also included is a chapter on climate, heliotherapy, mineral springs and health resorts. The discussion on hydrotherapy brings to the practitioner's attention the beneficial effects of heat and cold as a tonic, eliminative, sedative and stimulant. Harrington's work on chemistry of the thyroid principle, the use of dessicated swine stomach in pernicious anemia, salyrgan, postural rest in tuberculosis, as advocated by Webb, Calmette's B.C.G., tuberculin, undulant fever, ethylene, and the use of parathyroid hormone in Paget's disease are discussed.

Epidemiological Essays. By F. G. Crookshank, M.D., F.R.C.P. 136 pages. New York: The Macmillan Company, 1931. Cloth, $2.50.

The title of this book does not fully convey its contents, for it is a collection of contributions by the author to medical journals and papers presented before medical societies.

"Why Time Flies," the first chapter, is a philosophical consideration of time and, although not related to epidemiology, is well presented.

In chapter 2, "Airs, Waters, and Places," the author is too enthusiastic on spa treatment of disease.

In "Hot Waters" a brief statement is made of the benefits derived from this form of therapy and a detailed description is given to a little known French spa.

"Some Problems of Influenza," chapter 4, is an epidemiological treatise, but the breadth of the conception of the meaning of influenza held by the author exceeds the usual meaning of this disease.

"The Treatment of Encephalitis Lethargica" gives the author's opinions of the treatment of this disease, but is not related to the title of the book.

"The Trousse-Galants of 1528-29 and 1545-46" is written to prove that these epidemics were influenza, and from the data presented there is reason to accept his contention.

"Acrodynia" is written to prove that the disease described under this name was a post-influenzal manifestation. After reading this brief essay nothing definite seems to be established.

"Botulism and Heine-Medin Disease" is outstanding and was written to counteract a panic in Great Britain during the war. He shows that in many cases the disease was influenza misdiagnosed. The statement of the late Sir William Osler is worthy of mention: "Your paper is the most sensible thing written on this subject."

The concluding section, "The Defensive Value of Normal Mucus Formation" and "The Mortality of Pneumonia," are unrelated to epidemiology and are below the standard of the other contributions.

Nursing Technic. By Mary C. Wheeler, R. N., Former Superintendent of Illinois Training School for Nurses; Amalia Metzker, R. N., Instructor, St. Luke's Hospital School of Nursing, Racine, Wisconsin. Third Edition. Completely Revised and Reset, Illustrated. 333 pages. Philadelphia: J. B. Lippincott Company, 1930. Cloth, $1.75.

Each phase of nursing technic is detailed in synopsis form, the book being divided into four sections: housekeeping, general care of the patient, medical topics and surgical topics, respectively. It is a most complete and thorough text. Not only will the nurse find daily use for this book, but the physician will get many short cuts to the "Art of Medicine" from its pages.

Text-Book for Nurses—Anatomy, Physiology, Surgery and Medicine. Fourth Edition. By E. W. Hey Groves, M.D., B.Sc., M.S., F.R.C.S.; Surgeon to the Bristol General Hospital; Professor of Surgery, University of Bristol, and the Late J. M. Fortescue-Brickdale, M.A., M.D. (Oxon.), M.R.C.P. (Lond.), Senior Assistant Physician, Bristol Royal Infirmary. The Medical Section Revised by J. A. Nixon, C.M.G., M.D. (Cantab.), F.R.C.P. (Lond.), Professor of Medicine and Director of Clinical Medicine in the University of Bristol. 641 pages with illustrations. New York: The Oxford University Press, 1930.

Not a great amount of new material has been included in this new edition, except in the section on medicine. Here many alterations have been made in those chapters on specific infectious diseases and infections of the nervous system. The previous edition contained a chapter on war surgery. This has been omitted, parts of the chapter being retained elsewhere in the text. The book is comprehensive, covering anatomy and physiology, surgery and medicine. Its simplicity and accuracy should appeal to the nurse.

Southern Medical News

PAN-AMERICAN MEDICAL CONGRESS

The Third Congress of the Pan-American Medical Association will be held at Mexico City, Mexico, under the auspices of the Government of the Republic of Mexico, July 26-31. A cordial invitation is extended to physicians interested in medical interchange among English, Spanish, French and Portuguese speaking physicians of Pan-America.

The conference meets for the purpose of maintaining and promoting a more intimate understanding between the medical men of the New World. The proceedings of these Congresses reveal a record of achievements so necessary for the better scientific knowledge among our countries and is a step towards international medical progress.

Dr. Francisco M. Fernandez, Minister of Health, Republic of Cuba, Havana, is President of the Association; Dr. Jose E. Lopez Silvero, Secretaria de Sanidad, Havana, is Executive Secretary; Dr. Conrad Berens, New York, is Treasurer; and Dr. Francisco de P. Miranda, Departmento de Salubridad, Mexico City, is Executive Secretary of the Congress.

MEDICAL CLINIC IN PARIS

A medical clinic will be held at the Hopital de la Charite, 47 Rue Jacob, Paris, from July 6 to July 11, 1931, under the direction of Professor Sergent, member of the Academy of Medicine. Ten lectures will be given on diseases of the lung, with x-ray projections and anatomical specimens, each lecture being followed by practical demonstrations by MM. Bordet, Turpin, Kourilsky, Benda, and Iselin, occupying the positions of former clinical chiefs and clinical chiefs of the clinic. In the mornings practical demonstrations will be held in the wards under the guidance of Professor Sergent.

ALABAMA

The Alabama State Medical Association will hold its sixty-fourth annual session in Birmingham April 21-24 at the Thomas Jefferson Hotel. Among the out-of-state speakers on the program will be Dr. R. S. Cunningham, Vanderbilt University School of Medicine, Nashville; Dr. Olin West, Secretary, American Medical Association, Chicago; Dr. Alfred Blalock, Nashville; Dr. Hines Roberts, Atlanta; Dr. W. A. Smith, Atlanta; Dr. Lucius Burch, Nashville; Dr. J. T. Cater, Chicago; and Dr. Joseph B. Greene, Asheville, N. C.

Lee County Medical Society has elected as officers for 1931: Dr. Byron S. Bruce, Opelika, President; Dr. Gilmer H. Moore, Opelika, Vice-President; Dr. O. L. Chason, Opelika, Secretary-Treasurer; and Dr. F. R. B. Coggin, Waverly, Member of the Board of Censors for a five-year term.

Edward A. Boyden, Ph.D., is leaving the University of Alabama School of Medicine to become Professor of Anatomy at the University of Minnesota School of Medicine.

Dr. W. T. Fales, Director, Bureau of Vital Statistics of the State Health Department, Montgomery, sailed in February for Roumania, where he was sent by the Rockefeller Foundation to cooperate with the Roumanian government in developing its vital statistics.

The Alabama Academy of Science held its eighth annual meeting March 13 and 14 at the University of Alabama. Officers elected for the year 1931 are: George Fertig, Birmingham, President; Herbert Martin, Auburn, First Vice-President; H. C. Heath, Montgomery, Second Vice-President; E. A. Hawk, Ensley, Treasurer; J. L. Brakefield, Birmingham, Permanent Secretary. The next annual meeting will be held at Howard College, Birmingham.

Deaths

Dr. William H. Godwin, Thomas, aged 46, died January 7 from gunshot wounds.

Dr. Charles Ives Pegues, Safford, aged 70, died January 12 in Florida of bronchopneumonia.

ARKANSAS

The Arkansas Hospital Association held a meeting in February at which more than twenty-five hospitals were represented. At this meeting Lee C. Gammill, Superintendent of the Baptist State Hospital, Little Rock, was elected President; Dr. Charles S. Holt, Fort Smith, was reelected Vice-President; Miss Carolyn Snyder, Superintendent of Trinity Hospital, Little Rock, was elected Secretary-Treasurer.

The Boone County Medical Society elected as officers Dr. W. T. Moore, Everton, President; Dr. D. L. Owens, Harrison, First Vice-President; Dr. J. G. Gladden, Western Grove, Second Vice-President; Dr. J. H. Fowler, Harrison, Third Vice-President; and Dr. W. H. Poynor, Harrison, Secretary-Treasurer.

Columbia County Medical Society met in January, when it elected the following officers: Dr. J. J. Baker, Magnolia, President; Dr. W. P. Cooksey, Magnolia, Vice-President; and Dr. G. F. McLeod, Magnolia, Secretary-Treasurer.

Garland County-Hot Springs Medical Society has elected the following officers: Dr. J. S. Stell, President; and Dr. G. A. Hebert, reelected Secretary.

Ouachita County Medical Society met February 5 and designated their meeting as "Ladies' Night."

Dr. A. J. Harrison has moved from Lowell to Springdale.

Dr. J. R. McDaniel, Jr., has moved from Blytheville to Las Vegas, Nevada.

Dr. C. T. Morris has moved from Blytheville to Shreveport, Louisiana.

Dr. W. J. Robinson, Portia, and Miss Belle Ketchem, Imboden, were married February 4.

Deaths

Dr. George Hilbaird Setzler, Crossett, aged 66, died of heart disease November 23, 1930.

Dr. Charles Eugene Swift, Elkins, aged 56, died January 10 of injuries received in an auto accident.

Dr. Nettie Klein, Texarkana, aged 66, died February 5 of cerebral hemorrhage.

DISTRICT OF COLUMBIA

Sister Rochus has been named Superintendent of Georgetown University Hospital at Washington.

Fr. Francis J. Eiseman has been appointed Superintendent of Garfield Hospital, Washington, to succeed Colonel D. C. Howard, resigned.

Deaths

Dr. Julius Evan Lewis, Washington, aged 31, died December 1 of heart disease while on a sea voyage.

Dr. Charles I. Griffith, Washington, aged 53, died December 21.

Dr. Elmond Arthur Burnham, Washington, aged 65, died February 11 of cerebral hemorrhage.

Dr. John P. Briscoe, Washington, aged 49, died February 15 of pneumonia.

FLORIDA

The medical staff of the Duval County Hospital, Jacksonville, recently presented the hospital with a portrait of Dr. John E. Boyd, who for many years was connected with the institution and worked untiringly for its development.

The Good Samaritan Hospital Staff, West Palm Beach, elected the following officers for 1931: Dr. R. O. Cooley, Chief of Staff; Dr. W. O. Arnold, Secretary of Staff; Dr. W. W. George, Chief, Medical Division; Dr. G. M. Dawson, Chief, Surgical Division; and Dr. Fred K. Herpel, Chief, Laboratory Division.

The Hillsboro County Medical Society has elected the following officers for 1931: Dr. C. A. Andrews, Tampa, President; Dr. George L. Cook, Tampa, Vice-President; Dr. J. T. Cowart, Tampa, Secretary-Treasurer; and Dr. W. C. Blake, Dr. A. M. C. Jobson, Dr. Bundy Allen, Dr. R. P. Henderson, Dr. B. W. Lowry, Delegates to House of Delegates of the State Association.

Continued on page 34

Use DRYCO
in Nutritional Anemia!

Iron Is a Vital Factor in Infant Metabolism

The proportion of mineral elements of the milk is an important factor in promoting basal metabolism and satisfactory feeding.

Babies Must Get their Iron from Milk!

The iron content of DRYCO is two to five times as great as that of natural liquid milk. "This increase in iron content is obviously due to the intimate contact of the milk with the desiccating cylinders." DRYCO, the roller-processed dried milk, "has failed to cause the degree of nutritional anemia commonly reported for natural liquid milk." (Supplee, G. C., Dow, O. D., Flanigan, G.E., and Kahlenberg, O.J., Journal of Nutrition, Vol. II, No. 5, May, 1930.)

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A continuous success since the first edition was published in 1924—seven years of outstanding service to the general practitioner of medicine

Modern Methods of Treatment

By Logan Clendening, M.D.

Professor of Clinical Medicine, Lecturer on Therapeutics, Medical Department of the University of Kansas; Attending Physician, Kansas City General Hospital; Physician to St. Luke's Hospital, Kansas City, Mo.

819 pages, 6½x9½, with 95 illustrations in the text, and a color plate. Price, silk cloth binding, $10.00.

WITH CHAPTERS ON SPECIAL SUBJECTS BY H. C. Anderson, M.D.; J. B. Cowherd, M.D.; H. P. Kuhn, M.D.; Carl O. Rickter, M.G.; F. C. Neff, M.D.; E. H. Skinner, M.D.; and E. R. DeWeese, M.D.

New Revised Fourth Edition

In every case the author has tried to keep in mind the problems of the general practitioner and to encourage him to use methods and to adopt methods which he is likely to believe are effective only in the hands of what the world has unfortunately come to call specialists. To make the books as practicable as possible the author selected illustrations that really guide the physicians who study them and the captions beneath them. These illustrations are in themselves a course in therapeutics.

This new fourth edition has been carefully revised and all new methods in therapy added. The general plan and purpose of the book, found so useful to practitioners of medicine, have not been changed. The aim of the author has been to describe each method of procedure so clearly and minutely that a person who has never seen it performed could do it from the description in the book.

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Address .....

(SMJ)

Continued from page 274

The Marion County Medical Society has elected Dr. E. G. Lindner, Ocala, President; Dr. J. N. Moore, Ocala, Vice-President; Dr. T. H. Wallis, Ocala, Secretary-Treasurer; and Dr. A. H. Freeman, Dr. E. G. Peek, Dr. H. C. Dozier, all of Ocala, Board of Censors.

Dr. Julius C. Davis, Quincy, President of the State Medical Association, recently paid an official visit to the Societies at Tallahassee, Lake City and Jacksonville.

Dr. G. H. Edwards, Orlando, Chairman of the Orlando Utilities Commission for 1931, has been reelected a member of the Commission for a four-year term.

Dr. W. N. Parkinson, Dean of the Medical School of Temple University, Philadelphia, visited St. Augustine recently, where he was formerly Chief Surgeon of the East Coast Hospital.

Dr. Robert M. Faver, Lake Geneva, is spending some time in the clinics of the Eye, Ear, Nose and Throat Hospital at Memphis, Tennessee.

Dr. W. Grady Page has returned to Jacksonville after two years as House Surgeon at the New York Eye and Ear Infirmary, New York, N. Y. He will be associated with Dr. H. Marshall Taylor and Dr. Shaler Richardson.

Dr. Herbert Caldwell has been transferred from the U. S. Veterans' Hospital, Lake City, to the medical service in the U. S. Veterans' Hospital in Chicago.

Deaths

Dr. Oscar H. Fleming, Crestview, aged 62, died January 21.

Dr. Minerva B. Cushman, St. Cloud, aged 74, died October 27, 1930, of heart disease.

Dr. Charles Robert Oglesby, Bartow, aged 79, died January 16 of heart disease.

GEORGIA

The second annual assembly of the Southeastern Surgical Congress was held in Atlanta March 9 and 10 at the Biltmore Hotel. A very complete and comprehensive two-day surgical program was presented, with a banquet one evening. The meeting was well attended. Dr. E. G. Ballenger, Atlanta, was President. Dr. C. W. Roberts, Atlanta, is the new President. Dr. Frank K. Boland, Atlanta, is President-Elect, and Dr. B. T. Beasley, Atlanta, is Secretary-Treasurer.

The following counties were placed on the 1931 honor roll for having all of their eligible physicians as members of the county society: Randolph, Butts, and Monroe.

Emanuel County Medical Society has elected Dr. S. S. Youmans, Oak Park, President; Dr. E. C. Powell, Swainsboro, Vice-President; and Dr. R. C. Franklin, Swainsboro, Secretary-Treasurer.

Jenkins County Medical Society has elected Dr. M. E. Perkins, Millen, President; Dr. H. G. Lee, Millen, Vice-President; and Dr. C. Thompson, Millen, Secretary-Treasurer.

Lowndes County Medical Society has elected Dr. G. T. Crozier, Valdosta, President; Dr. J. P. Prescott, Lake Park, Vice-President; and Dr. B. G. Owens, Valdosta, Secretary-Treasurer.

Macon County Medical Society has elected Dr. C. P. Savage, Montezuma, President; and Dr. T. M. Adams, Montezuma, Secretary-Treasurer.

Stewart-Webster Counties Medical Society has elected Dr. W. F. McCurdy, Weston, President; Dr. C. S. Lynch, Lumpkin, Vice-President; and Dr. J. M. Kenyon, Richland, Secretary-Treasurer.

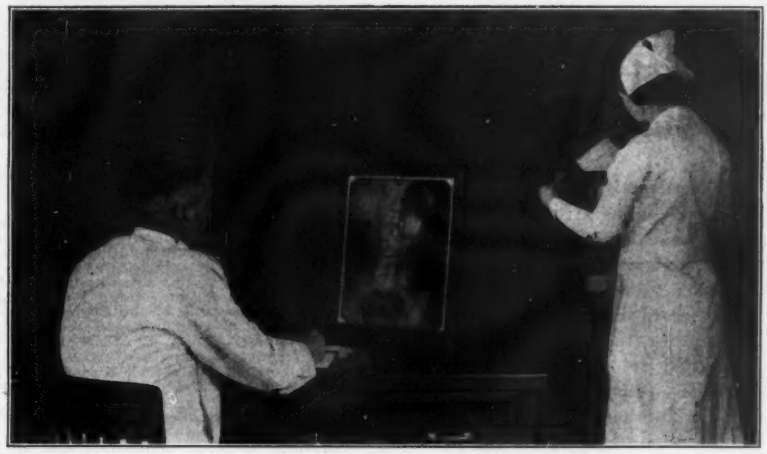
Taylor County Medical Society has elected Dr. W. W. Edwards, Butler, President; Dr. S. H. Bryan, Reynolds, Vice-President; and Dr. R. C. Montgomery, Butler, Secretary-Treasurer.

Thomas County Medical Society has elected Dr. L. L. Lundy, Boston, President; Dr. Harry Ainsworth, Thomasville, Vice-President; and Dr. C. J. Reilly, Thomasville, Secretary-Treasurer.

Turner County Medical Society has elected Dr. W. L. Story, Ashburn, President; Dr. F. W. Rogers, Ashburn, Vice-President; and Dr. J. H. Baxter, Ashburn, Secretary-Treasurer.

Walker County Medical Society has elected Dr. D.

Continued on page 36



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For a quarter hour of stimulating entertainment, tune in on "Devils, Drugs, and Doctors," broadcast each Sunday evening at 8 o'clock New York time, over a coast-to-coast network of the Columbia System. These talks, sponsored by Eastman Kodak Company, are given by Dr. Howard W. Haggard, Associate Professor of Applied Physiology, Yale University.

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THE advertisement reproduced here is one of the series appearing in newspapers throughout the country. In order to keep the statements in accord with modern medical practice, they have been submitted to and approved by some of the leading authorities in the field of human nutrition in the United States. The Sugar Institute, 129 Front St., New York.

Between-meal fatigue is a signal
to eat
something
sweet
Thousands of workers and shoppers overcome fatigue in mid-afternoon by eating a food or drinking a beverage with sugar in it.
It is unwise to endure fatigue. For fatigue may develop into exhaustion, and when the system is in that state, it has virtually no resistance. Colds and common ills are inevitable.
It is a good idea when you feel fatigued, to stop a moment and eat or drink something sweet. Such foods or beverages are quickly digested—the body is nourished, and the energy in the sugar is ready at once to renew vigor.
Appropriate food for sweet snacks can be had in soda fountain beverages, ice cream, candy and small cakes. For children coming home from school, sweetened and flavored milk shakes, also bread and butter sprinkled with sugar are recommended. Most foods are more delicious and nourishing with sugar.
The Sugar Institute.
"A flavored drink is refreshing"

Continued from page 34

W. Hammond, Lafayette, President; Dr. S. B. Kitchen, Lafayette, Vice-President; and Dr. J. H. Hammond, Lafayette, Secretary-Treasurer.

Warren County Medical Society has elected Dr. H. B. Cason, Jr., Warrenton, President; Dr. H. T. Kennedy, Warrenton, Vice-President; and Dr. A. W. Davis, Warrenton, Secretary-Treasurer.

Wayne County Medical Society has elected Dr. J. Lawton Tyre, Screven, President; Dr. I. K. Ogden, Odum, Vice-President; and Dr. A. J. Gordon, Jesup, Secretary-Treasurer.

Whitfield County Medical Society has elected Dr. J. C. Rollins, Dalton, President; Dr. Frank Easley, Dalton, Vice-President; and Dr. E. O. Shellhorse, Dalton, Secretary-Treasurer.

Wilcox County Medical Society has elected Dr. R. A. Williams, Abbeville, President; and Dr. W. R. Googe, Abbeville, Secretary-Treasurer.

The Georgia State Board of Health has added a new unit to its field equipment for fighting tuberculosis known as the "X-Ray Mobile." It is an automobile equipped with a complete x-ray outfit.

Dr. Frank W. Wells, Atlanta, is the newly elected President of the Fulton County Board of Health.

The annual meeting of the Physicians and Surgeons Association of the Georgia and Florida Railroad was held in January, Dr. A. S. M. Coleman, Douglas, presiding.

Dr. Robert M. Ware and Miss Sarah Pollard, both of Fitzgerald, were married January 6 in Lake City, Florida.

Dr. Samuel Rosen, Savannah, and Miss Estelle Sawilowski, Augusta, were married December 14.

Dr. John W. Good, Cedartown, and Miss Lois Bedwell, Cedar Bluff, Alabama, were married December 5.

Deaths

Dr. James A. Ward, Cordele, aged 62, died January 10 at his home.

Dr. Edward B. Terrell, Greenville, aged 73, died January 12 in an Atlanta hospital.

Dr. William Holt Worrill, Ochlochnee, aged 48, died January 15 at his home.

Dr. Captain Hunter House, Atlanta, aged 48, died January 15 of pneumonia.

Dr. Percival Ranney Bolton, Thomasville, aged 65, died February 4 of angina pectoris.

Dr. Samuel John Wiley, Columbus, aged 67, died January 20 of acute dilatation of the heart.

Dr. James Nimmo Ellis, Atlanta, aged 67, died February 11.

KENTUCKY

The Barren County Medical Society has elected Dr. Clifton Richards, Glasgow, President; Dr. Paul S. York, Vice-President; and Dr. C. C. Turner, Glasgow, Secretary-Treasurer.

The Clark County Medical Society held its annual dinner and election of officers recently. The following were elected: Dr. Robert H. Scobee, Winchester, President; Dr. John A. Snowden, Sr., Winchester, Vice-President; and Dr. George F. Doyle, Winchester, Secretary-Treasurer.

The McCracken County Medical Society has elected Dr. Errett Pace, Paducah, President; Dr. Bob Overbey, Paducah, Vice-President; Dr. J. T. Reddick, Paducah, Secretary; and Dr. P. H. Stewart, Paducah, Treasurer (reelected).

The Kentucky State Medical Association, with the co-operation of the Faculty of the University of Louisville, will again this year hold a post-graduate course. Lectures will be held June 1-3.

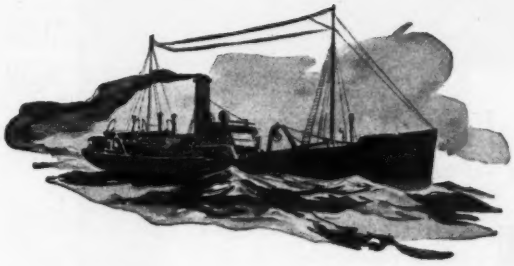
Dr. J. W. Davis, Hazard, has been elected Health Officer of Fayette County for the remainder of the unexpired term. Dr. Rollin E. May, Whitesburg, who held this office, resigned January 1.

Dr. James S. Lock, Barbourville, has tendered his resignation as Secretary of the Kentucky Tuberculosis Association.

Dr. John B. Overall and Miss Elizabeth Waters, both of Springfield, were married December 31.

Dr. Logan Felts and Miss Ariena Dillon, both of Russellville, were married in Marion January 27.

Continued on page 38



The Pastures of the Sea

Where Patch begins the manufacture of cod liver oil is out on the fenceless meadows where the fish are caught.

It was Patch who invented the cooker which makes possible the extraction of oil from the livers of fresh codfish. Every step—from fish to finished product—is carefully controlled by Patch experts, to insure an oil of maximum vitamin potency and unusual palatability.

The Patch guaranty below means something to you, doctor. See what a rich vitamin A and D potency you are assured when you prescribe Patch's Flavored Cod Liver Oil.

As to palatability—well, the best demonstration of that is a taste of the oil itself. The coupon below brings you a bottle for actual manifestation of this important factor.

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1000 A Units per gram of oil
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Gentlemen: Please send me a sample of Patch's Flavored Cod Liver Oil and literature.

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Continued from page 36

Deaths

Dr. Tilman Johnson Robb, North Fork, aged 49, died January 9 from pneumonia.

Dr. George Nathaniel Murphey, Paducah, aged 82, died January 13 from heart disease.

LOUISIANA

Beauregard Parish Medical Society has elected Dr. Thomas A. Guillory, Merryville, President; Dr. J. D. Frazar, DeRidder, Vice-President; and Dr. S. O. Turner, DeRidder, Secretary-Treasurer.

In using lactic acid milk for infant feeding, physicians find Mead's Powdered Non-curdling Lactic Acid Milk No. 1 (containing Dextri-Maltose) the simplest and most satisfactory medium for the preparation of lactic acid milk in the home or hospital. The proper amount of carbohydrate incorporated in this product correlates the lactic acid content, so that it is always ready for use. This product never curdles. Samples and literature sent on request. Mead Johnson & Company, Evansville, Ind., U.S.A.

Franklin Parish Medical Society has elected Dr. A. J. Reynolds, Winnsboro, President; Dr. R. L. Segrest, Wisner, Vice-President; and Dr. R. E. King, Treasurer.

Morehouse Parish Medical Society has elected Dr. R. B. Leavel, Bastrop, President; Dr. E. L. Miller, Bonita, Vice-President; and Dr. Smith I. Sims, Bastrop, Secretary-Treasurer.

St. Landry Parish Medical Society has elected Dr. L. J. Bienville, Opelousas, President; Dr. J. N. Brown, Washington, Vice-President; and Dr. W. R. Lastrapes, Opelousas, Secretary-Treasurer.

Deaths

Dr. Thomas Ragan, Shreveport, aged 64, died January 9 of cerebral arteriosclerosis and cerebral edema.

Dr. Elisha W. Breazeale, Campti, aged 61, died January 13 from injuries received in an auto accident.

Dr. Thomas W. Breaux, New Orleans, aged 58, died January 17 of chronic myocarditis.

MARYLAND

Dr. Wade Hampton Frost, Baltimore, has been appointed Dean of Johns Hopkins School of Hygiene and Public Health to succeed Dr. William H. Howell, retiring. Dr. Frost will take office July 1. He was formerly Professor of Epidemiology at Johns Hopkins.

Dr. Thomas W. Koon, Cumberland, and Miss Fannie Hutson, Rawlings, were married November 12.

Deaths

Dr. Charles Hill Brooke, Baltimore, aged 64, died January 28 of influenza and internal hemorrhage.

Dr. Ernest Luther Bullard, Rockville, aged 71, died January 23 of dilatation of the heart.

Dr. John William Schmidt, Brookview, aged 82, died in October.

Dr. Frank August Sigrist, Baltimore, aged 36, died January 27 of lobar pneumonia.

MISSISSIPPI

The Edwards Hotel, Jackson, has been designated as headquarters for the annual meeting of the State Medical Association in May. The following men were named as a committee on arrangements: Dr. H. F. Garrison, Reception; Dr. A. G. Wilde, Local Arrangements; Dr. Willis Walley, Hospitals and Clinics; Dr. A. E. Gordin, Entertainment; and Dr. N. C. Womack, Finance.

Tate County Medical Society has elected Dr. H. F. Byers, Senatobia, President; Dr. William R. Gilbert, Tyro, Vice-President; and Dr. J. Sidney Eason, Coldwater, Secretary-Treasurer.

Dr. A. C. Bryan and Dr. K. T. Klein announce that they have taken over the management of the Meridian Sanitarium. Dr. Klein limits his practice to general surgery.

Dr. H. Lowry Rush and Dr. Leslie V. Rush, Directors of Rush's Infirmary, Meridian, have announced the limitation of their practice to surgery and consultations.

Anderson Infirmary, Meridian, has a new fifteen-room annex fitted with the latest hospital equipment, which includes an electric elevator and an automatic coal burner heating apparatus.

Dr. Julian T. Bailey, Meridian, and Miss Mary Stella Mitchell were married in Meridian January 7.

Dr. Ellis Columbus Moore, Carthage, and Miss Catherine Preston Finney, Lexington, Virginia, were married December, 1930.

Deaths

Dr. J. H. Rush, Meridian, aged 62, died January 22 after a brief illness.

Dr. E. J. Rowe, Hillsboro, aged 69, died January 7 of apoplexy.

Dr. C. T. Bell, DeKalb, aged 52, died January 26.

Dr. R. C. Basinger, Jackson, aged 31, died January 31 of cerebral hemorrhage.

Dr. Tillis Gandy, Royce, aged 45, died December 26 of pneumonia.

Dr. Bolivar R. Smith, Prentiss County, aged 80, died December 9.

Continued on page 40

Patient Types . . .

The Chronic

They have worn holes in the carpets of many a waiting room and frayed the physicians' patience to shreds.

Often, underlying the chronic condition is bowel stasis and irrational use of harsh cathartics.

In such cases many chronics have been definitely benefited by a period of "habit time" education together with other rational treatment.

The use of Petrolagar will materially shorten the period of bowel re-education. A few of the advantages of using Petrolagar over plain mineral oil are its palatability, its more thorough permeation of the feces, less danger of leakage, and it has no deleterious effect on digestion.

Petrolagar

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Gentlemen: — Send me copy of the new brochure "HABIT TIME" (of bowel movement) and specimens of Petrolagar.

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The results of twenty years' clinical experience in the foremost medical centers of this country, are your precedent for prescribing

OLEO-GOMENOL

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CYSTITIS

℞ 5 to 15cc, by injection direct into the bladder.

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Nitrous Oxide
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For safety reasons we differentiate our gases with distinctive colors over the entire cylinder, as recommended by resolution of the International Anesthesia Research Society.

Puritan Compressed Gas Corp.
Formerly Kansas City Oxygen Gas Co.

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ST. LOUIS, MO. 4578 Laclede Ave.
CINCINNATI, OHIO 6th and Bay Miller Sts.

Latest Oxygen Tent Therapy Equipment

Continued from page 38

MISSOURI

Dr. Walter Baumgarten, St. Louis, has been appointed the member of the Council from Missouri of the Southern Medical Association, the appointment having been announced recently by the President, Dr. Felix J. Underwood, Jackson, Mississippi. Dr. Baumgarten succeeds Dr. W. McKim Marriott, whose term has expired, and having served the constitutional limit, was not eligibile to reappointment.

Franklin County Medical Society has elected Dr. W. E. Kitchell, St. Clair, President; and Dr. H. A. May, Washington, Secretary-Treasurer.

Johnson County Medical Society has elected Dr. J. A. Powers, Warrensburg, President; Dr. Ralph McKenny, Warrensburg, Vice-President; and Dr. T. J. Draper, Warrensburg, reelected Secretary-Treasurer.

Lawrence-Stone County Medical Society has elected Dr. E. E. Glenn, Mt. Vernon, President; Dr. J. C. R. Doggett, Crane, Vice-President; and Dr. R. D. Cowan, Aurora, Secretary-Treasurer.

Platte County Medical Society has elected Dr. Wilson Murray, Platte City, President; Dr. J. M. Hale, Dearborn, Vice-President; and Dr. Spence Redman, Platte City, Secretary-Treasurer.

St. Charles County Medical Society has elected Dr. Vincent A. Schneider, St. Charles, President; Dr. B. Geret Gossow, St. Charles, Vice-President; and Dr. D. L. E. Belding, St. Charles, Secretary-Treasurer.

Ste. Genevieve County Medical Society has elected Dr. J. A. Wilkins, St. Marys, President; Dr. G. M. Rutledge, Ste. Genevieve, Vice-President; and Dr. R. W. Lanning, Ste. Genevieve, Secretary-Treasurer.

Dr. G. Wilse Robinson, Kansas City, recently opened a school for defective children which is called the "Robinson School for Different Children." Medical supervision is given all types of subnormal children.

At the annual meeting of the Missouri State Board of Health Dr. Francis M. McCallum, Kansas City, was elected President; Dr. Horace W. Carle, St. Joseph, Vice-President; and Dr. James Stewart, Jefferson City, reelected Secretary.

Dr. Charles T. Reid. Joplin, has gone to Vienna to attend post-graduate clinics in diseases of the eye.

The Missouri-Pacific Hospital Association elected Dr. Emmett P. North, St. Louis, President; Dr. James R. McVay, Kansas City, Vice-President; and Dr. Henry J. Schenck, St. Louis, Secretary.

Ann Kersting, Poplar Bluff, has been appointed Superintendent of the Poplar Bluff Hospital.

Deaths

Dr. Waller J. Parker, Steelville, aged 71, died December 1.

Dr. Michael Golland, St. Louis, aged 68, died January 17.

Dr. Arthur Campbell Kimball, St. Louis, aged 52, died February 3.

Dr. Edwin A. Mendeel, St. Joseph, aged 69, died January 14.

Dr. David Wise, Carthage, aged 61, died December 9, 1930.

Dr. Tinsley Brown, Hamilton, aged 81, died February 9.

Dr. Solomon R. McKay, Troy, aged 76, died January 5 of arteriosclerosis and diabetes mellitus.

Dr. Andrew H. Starcke, Kansas City, aged 56, died February 3 in the Evangelical Hospital.

Dr. Franklin George Johnson, Sheridan, aged 57, died January 6 of infection of the spinal cord and paralysis.

Dr. Henry Westren Miller, Kansas City, aged 85, died in December.

NORTH CAROLINA

The new Duke Hospital at Durham has named fourteen of its new wards after prominent men in the medical history of the South.

The Lowrance Hospital at Mooresville, erected at a cost of $125,000, was recently opened. The Duke Hospital Foundation donated $10,000 for the building.

Dr. James W. Davis, Statesville, and Miss Nancy Smith, Greensboro, were married December 10.

Continued on page 42

The Well Baby Eats Well

•

When the Supplementary Feeding is KLIM Powdered Whole Milk!

Breast-fed **babies who are not making satisfactory gains and receiving insufficient nourishment should receive supplementary feedings of KLIM powdered whole milk.**

Bottle-fed **babies should receive, at regular intervals, all the milk they need to satisfy their appetites and to insure the growth curve.**

Clinical experience **has shown that babies make more satisfactory gains in growth and have a higher resistance to infection and disease when fed whole milk which is uniform in its constituents and is easily digested. Klim is such a milk.**

"The giving of food of too low a caloric value to meet the infant's needs is by all odds the chief cause of failure in infant feeding." **Marriott, Infant Nutrition, Mosby, 1930.**

ACCEPTED AMERICAN MEDICAL ASSN. Committee on Foods

(Recognizing the importance of scientific control, all contact with the laity is predicated on the policy that Merrell-Soule products be used in infant feeding only according to a physician's formula.)

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KLIM

POWDERED WHOLE MILK

INSURE THE WEIGHT CURVE ~ *PRESCRIBE* KLIM

Continued from page 40

Dr. Williamson Ziegler Bradford and Miss Mary Coley Spier, both of Charlotte, were married January 6.

Dr. George Britain Walton, Durham, and Miss Liza Witherspoon, Greensboro, were married in Winston-Salem December 12.

OKLAHOMA

The former Oklahoma Hospital at Tulsa will be reopened shortly under the management of Dr. Ned R. Smith, Tulsa. It will be devoted to the treatment of nervous and mental cases.

Dr. George N. Bilby, Alva, has been made State Health Commissioner, succeeding Dr. Clyde W. Beson.

At the January meeting of the Adair County Medical Society the following officers were elected for 1931: Dr. I. W. Rogers, Watts, President; Dr. R. M. Church, Stilwell, Vice-President; and Dr. D. P. Chambers, Stilwell, Secretary.

Officers for the Alfalfa County Medical Society for 1931 are: Dr. Z. J. Clark, Cherokee, President; Dr. J. W. Mercer, Cherokee, Vice-President; and Dr. L. T. Lancaster, Cherokee, Secretary-Treasurer.

Caddo County Medical Society has elected Dr. J. H. Cantrell, Carnegie, President; and Dr. P. H. Anderson, Anadarko, Secretary.

Canadian County Medical Society has elected officers for 1931 as follows: Dr. William J. Muzy, El Reno, President; Dr. H. A. Dever, El Reno, Vice-President; and Dr. A. L. Johnson, El Reno, Secretary-Treasurer.

Carter County Medical Society has elected Dr. Robert H. Henry, Ardmore, President; Dr. David Autry, Marietta, Vice-President; and Dr. R. C. Sullivan, Ardmore, Secretary-Treasurer.

Craig County Medical Society has elected Dr. D. B. Stough, Vinita, President; Dr. J. F. Walker, Grove, Vice-President; and Dr. P. L. Hays, Vinita, Secretary-Treasurer.

Pathologic Tissue Diagnosis

We are prepared to make routine microscopic examinations of tissues for smaller hospitals and clinics which do not employ a fulltime pathologist.

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St. Louis, Mo.

Custer County Medical Society has elected Dr. Lealon E. Lamb, Clinton, President; Dr. E. M. Loyd, Taloga, Vice-President; and Dr. E. E. Darnell, Clinton, Secretary-Treasurer.

Jefferson County Medical Society has elected Dr. F. M. Mingus, Ringling, President; and Dr. W. C. Burgess, Ringling, Secretary.

Kiowa County Medical Society has elected Dr. A. H. Hathaway, Mt. View, President; Dr. J. L. Adams, Hobart, Vice-President; and Dr. J. H. Moore, Hobart, Secretary-Treasurer.

McClain County Medical Society has elected Dr. I. N. Kolb, Blanchard, President; Dr. W. C. McCurdy, Purcell, Vice-President; and Dr. J. H. Moore, Hobart, Secretary-Treasurer.

Murray County Medical Society has elected Dr. O. W. Sprouse, Sulphur, President; and Dr. H. C. Bailey, Sulphur, Secretary.

Noble County Medical Society has elected Dr. B. A. Owen, Perry, President; and Dr. J. W. Francis, Perry, Secretary-Treasurer.

Oklahoma County Medical Society has elected Dr. J. M. Alford, Oklahoma City, President; Dr. Hugh Jeter, Oklahoma City, Vice-President; and Dr. K. G. Parks, Oklahoma City, Secretary.

Okmulgee County Medical Society has elected Dr. W. S. Watson, Okmulgee, President; Dr. T. C. Carloss, Morris, Vice-President; and Dr. M. B. Glismann, Okmulgee, Secretary-Treasurer.

Osage County Medical Society has elected Dr. M. Karasek, Shidler, President; Dr. Roscoe Walker, Pawhuska, Vice-President; and Dr. M. E. Rust, Pawhuska, Secretary-Treasurer.

Pottawatomie County Medical Society has elected Dr. John I. Gaston, Shawnee, President; Dr. W. M. Gallaher, Shawnee, First Vice-President; Dr. R. M. C. Hill, Second Vice-President; and Dr. F. C. Gallaher, Shawnee, Secretary-Treasurer.

Rogers County Medical Society has elected Dr. A. M. Arnold, Claremore, President; Dr. J. C. Bushyhead, Claremore, Vice-President; and Dr. W. A. Howard, Chelsea, Secretary-Treasurer.

Seminole County Medical Society has elected Dr. T. H. Briggs, Wewoka, President; Dr. O. C. Butler, Seminole, Vice-President; and Dr. H. A. Kiles, Konawa, Secretary-Treasurer.

St. Anthony's Clinical Society, Oklahoma City, has elected Dr. W. K. West, Oklahoma City, President; Dr. N. P. Eley, Oklahoma City, Vice-President; and Dr. K. G. Parks, Oklahoma City, Secretary-Treasurer.

Deaths

Dr. Erastus Thomas Barker, Oklahoma City, aged 60, died October 16 in the U. S. Veterans' Hospital, North Little Rock, of cerebral hemorrhage.

SOUTH CAROLINA

At a recent meeting of the Anderson County Medical Society President-Elect, Dr. Frank Lander, Williamston, presented the Association with an historic gavel made of wood from a chair which had been in the home of Dr. Benjamin Franklin Brown, one of South Carolina's earliest medical men and the first male baby born in Anderson County. He was born in 1833, died in 1919. This incident was just one of many recently which have as their motive the bringing to general knowledge and the preservation of historic facts about the medical men of South Carolina.

Dr. Julian P. Price and Miss Caira Haynsworth, both of Florence, were married February 7.

Dr. Clifford Lee Welbourne and Miss Lottie Mae Webber, both of Charleston, were married January 20.

Deaths

Dr. John Merceir Green, Charleston, aged 61, died January 14 of arterioscierosis and angina pectoris.

Dr. Isaac Johnston Campbell, Clover, aged 49, died suddenly February 7 of angina pectoris.

TENNESSEE

The Tri-States Medical Association of Tennessee,

Continued on page 44

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Continued from page 42

Mississippi and Arkansas held its forty-seventh annual assembly in Memphis February 17-20 at the Peabody Hotel. A very comprehensive four-day general medical and surgical program was presented, with a banquet one evening and round table luncheon meetings each noon. The name was changed to the Mid-South Post-Graduate Assembly. Dr. N. S. Walker, Dyersburg, Tennessee, was the retiring President, Dr. A. G. Payne, Greenville, Mississippi, is the incoming President, and Dr. A. F. Cooper, Memphis, was re-elected Secretary.

Dr. Morgan Clack, formerly of Rockwood, is now associated with Drs. W. F. and W. A. Thomison in Dayton.

Aileen Brown, Fayetteville, is now Superintendent of Lincoln Hospital, Stanford, Kentucky.

Bradley County Medical Society has elected Dr. W. B. Campbell, Cleveland, President; Dr. R. O. Kibler, Cleveland, Vice-President; and Dr. S. J. Sullivan, Cleveland, Secretary-Treasurer.

Cumberland County Medical Society has elected Dr. E. W. Mitchell, Crossville, President; and Dr. V. L. Lewis, Crossville, Secretary-Treasurer.

Davidson County Medical Society has elected Dr. L. W. Edwards, Nashville, President; Dr. C. M. Hamilton, Nashville, Vice-President; and Dr. N. S. Shofner, Nashville, Secretary-Treasurer.

Dickson County Medical Society has elected Dr. L. F. Loggins, Charlotte, President; Dr. W. M. Cunningham, Cumberland Furnace, Vice-President; and Dr. W. J. Sugg, Dickson, Secretary.

Gibson County Medical Society has elected Dr. G. C. Bryant, Milan, President; Dr. B. S. Penn, Humboldt, Vice-President; and Dr. F. L. Roberts, Trenton, Secretary.

Henry County Medical Society has elected Dr. R.

Continued on page 46

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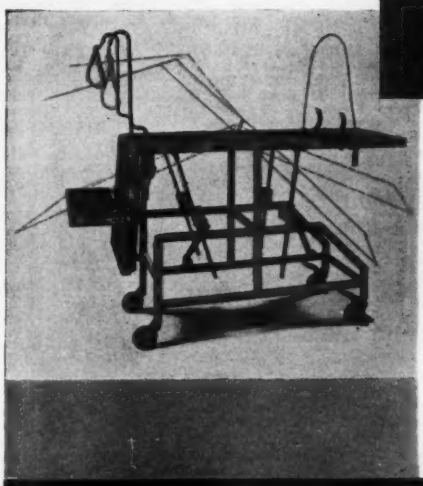

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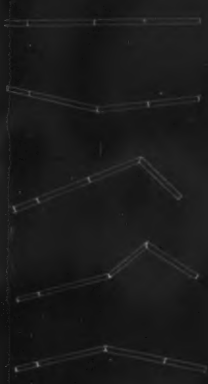

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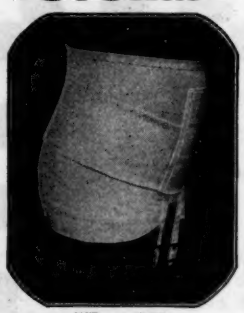

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Continued from page 44

G. Fish, Paris, President; Dr. George D. Boone, Paris, Vice-President; and Dr. A. A. Oliver, Paris, Secretary.

Hickman County Medical Society has elected Dr. John S. Beasley, Centerville, President; and Dr. W. K. Edwards, Centerville, Secretary.

Knox County Medical Society has elected Dr. Tom R. Barry, Knoxville, President; Dr. R. G. Waterhouse, Knoxville, Vice-President; and Dr. Jesse C. Hill, Knoxville, Secretary-Treasurer.

Sumner County Medical Society has elected Dr. Geoffrey Morris, Gallatin, President; Dr. J. M. Oliver, Portland, Vice-President; and Dr. W. M. Lackey, Gallatin, Secretary.

Dr. Robert C. Anderson, Johnson City, and Miss Thane Hendrix, Petersburg, Virginia, were married November 8 in Bristol, Virginia.

Deaths

Dr. Walter Fenner Rochelle, Jackson, aged 73, died January 9.

Dr. John Quincey Holifield, Tiptonville, aged 36, died January 22 in Memphis.

TEXAS

Dr. Witten B. Russ, San Antonio, is the newly elected President of the Texas Surgical Society.

Jewell Lovelace is now Superintendent of the Dr. J. C. McLean Hospital and Clinic, Pampa.

Bowie County Medical Society has elected Dr. J. N. White, Texarkana, President; Dr. Joe Tyson, Texarkana, First Vice-President; Dr. J. T. Robison, Texarkana, Second Vice-President; and Dr. A. W. Roberts, Texarkana, Secretary-Treasurer.

Officers for Brown County Medical Society for 1931 are: Dr. T. B. Bailey, President; Dr. W. H. Paige, Vice-President; and Dr. Earle B. Jones, Secretary-Treasurer, all of Brownwood.

Childress-Collingsworth-Donley-Hall-Wheeler Counties Medical Society has elected the following officers for 1931: Dr. H. S. Townsend, Childress, President; Dr. T. M. Morgan, Childress, Dr. B. L. Jenkins, Clarendon, Dr. C. E. High, Wellington, Dr. J. W. Beach, Shamrock, and Dr. Winfred Wilson, Memphis, Vice-Presidents; and Dr. P. R. Jeter, Childress, Secretary-Treasurer.

Dallas County Medical Society has elected Dr. C. M. Grigsby, Dallas, President; Dr. R. A. Trumbull, Dallas, Vice-President; and Dr. W. W. Fowler, Dallas, Secretary-Treasurer (reelected).

El Paso County Medical Society has elected Dr. J. W. Laws, El Paso, President; Dr. Steve A. Schuster, El Paso, Vice-President; and Dr. Ralph Homan, El Paso, Secretary-Treasurer.

Falls County Medical Society has elected Dr. J. H. Mitchell, Kosse, President; Dr. B. M. Avent, Rosebud, Vice-President; and Dr. J. I. Collier, Marlin, Secretary-Treasurer (reelected).

Fannin County Medical Society has elected Dr. H. H. Leeman, Windom, President; Dr. C. C. Adair, Bailey, Vice-President; and Dr. L. C. Biggers, Secretary-Treasurer (reelected).

Hardeman-Cottle Counties Medical Society has elected Dr. J. J. Hanna, Quanah, President; Dr. J. W. Conley, Quanah, Vice-President (reelected); and Dr. Mildred V. Hanna, Crowell, Secretary-Treasurer.

Harris County Medical Society has elected Dr. Fred R. Lummis, Houston, President; Dr. Joseph D. Walker, Houston, Vice-President; Dr. Byron P. York, Houston, Secretary; and Dr. J. C. Alexander, Houston, Treasurer.

Hill County Medical Society has elected Dr. L. D. Robertson, Malone, President; Dr. J. W. Miller, Hillsboro, Vice-President; and Dr. James E. Boyd, Hillsboro, Secretary-Treasurer (reelected).

Medina - Uvalde-Maverick-Valverde-Edwards-R-K-Z Counties Medical Society has elected Dr. Cary A. Poindexter, Crystal City, President; Dr. D. A. York, Del Rio, Vice-President; and Dr. W. P. Meredith, Del Rio, Secretary-Treasurer.

Navarro County Medical Society has elected Dr. W. C. Bristow, Emhouse, President; Dr. H. R. McMullan, Roane, Vice-President; and Dr. W. T. Shell, Jr., Corsicana, Secretary-Treasurer.

Continued on page 48

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Continued from page 46

Nueces County Medical Society has elected Dr. C. P. Yeager, Corpus Christi, President; Dr. O. H. Peterson, Corpus Christi, Vice-President; and Dr. C. P. Jasperson, Corpus Christi, Secretary-Treasurer (re-elected).

Parker County Medical Society has elected Dr. W. J. Sparks, Poolville, President; Dr. N. E. Dick, Millsap, Vice-President; and Dr. Alexander S. Garrett, Weatherford, Secretary (re-elected).

Webb County Medical Society has elected Dr. J. L. Crawford, Laredo, President; Dr. W. R. Powell, Laredo, Vice-President; and Dr. V. L. Puig, Jr., Secretary-Treasurer.

Wichita County Medical Society has elected Dr. F. R. Collard, Wichita Falls, President; Dr. O. C. Egdorf, Burkburnett, Vice-President; and Dr. J. A. Little, Wichita Falls, Secretary-Treasurer.

Williamson County Medical Society has elected Dr. C. R. Miller, Leander, President; Dr. B. A. Kirkpatrick, Thrall, Vice-President; and Dr. Van C. Tipton, Georgetown, Secretary-Treasurer (reelected).

Deaths

Dr. William Moody Cole, Longview, aged 66, died January 6 of cerebral hemorrhage.

Dr. Samuel W. Hart, Mineola, aged 65, died November 24.

Dr. James Abstone Miller, McAllen, died December 20 of pneumonia.

Dr. Thomas Buchanan Sadler, Corsicana, aged 61, died of heart disease November 13.

Dr. Stephen A. Foote, Houston, aged 60, died January 7 of coronary thrombosis.

Dr. Paul M. Donigan, Brookshire, aged 71, died in a Houston hospital December 27.

Dr. Frances May McAdams, Bryan, aged 45, died of heart disease January 6.

Dr. Martin L. Harrell, Dallas, aged 69, died December 6.

Dr. Jasper Halpenny, Harlingen, aged 61, died December 19 of pneumonia.

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Dr. Herbert Anderson White, Raymondville, aged 61, died January 24 of septicemia.

Dr. William Thomas Stokes, Alpine, aged 53, died January 25 of angina pectoris.

Dr. William Herman Anderson, Littlefield, aged 73, died November 11.

VIRGINIA

In the March issue in this column an item was carried to the effect that Dr. Frederick M. Allen had been made Professor of Internal Medicine and Dr Everett M. Hawks, Professor of Gynecology and Obstetrics in the University of Virginia Department of Medicine, University. This item was in error. The college connection of the physicians named is the New York Polyclinic Medical School and Hospital, New York. The JOURNAL regrets the error.

Dr. J. Belmont Woodson, Lowesville, has succeeded Dr. W. H. Venable, resigned, as Superintendent and Medical Director of the Piedmont Sanatorium, Burkeville, the state aided institution for negroes suffering from tuberculosis.

Lieutenant Charles L. Baird, Buckingham County, and Lieutenant Samuel L. Cooke, Pittsylvania County, both graduates of the Medical College of Virginia, were recently graduated from the Army Medical School in Washington.

Dr. William Edward Fitch, formerly of Buffalo Lithia Springs, has recently accepted the position as Consulting Medical Hydrologist and Medical Director of the French Lick Spa, French Lick, Indiana.

Dr. Peter P. Causey, Sadley, has succeeded Dr. Bathurst B. Bagby as Health Officer of Southampton County. Dr. Bagby has been promoted to Director of the Child Health Bureau of the State Board of Health, Richmond.

Dr. Nelson Mercer, Richmond, and Miss Jeannette Bahn, of Maryland, were married in Washington December 8.

Dr. Caleb S. Stone, Farmville, and Miss Margaret S. Carter, University, were married January 31.

Dr. William E. Chapin, Richmond, and Miss Nancy Berry, Prince George County, were married recently.

Dr. William Latimer Cooke, of the class of 1929, Medical College of Virginia, and Miss Sally Ann Boxley, Roanoke, were married February 10.

Dr. John Claiborne Palmer, Guinea, and Miss Jane Foster, The Plains, were married January 3.

Dr. Blake Walden Meador and Miss Ethel Marchant Hale, both of Richmond, were married December, 1930.

Deaths

Dr. C. F. Rinker, Upperville, aged 71, died November 11 of pneumonia.

Dr. William M. Carter, Keokee, aged 66, died October 16.

Dr. Sampson Busby Allen, Charlottesville, aged 77, died February 4 of cerebral arteriosclerosis.

WEST VIRGINIA

Dr. Guy Hinsdale has returned to White Sulphur Springs, where he is Medical Director, after a three months' stay in London and Paris.

The new Beckley Hospital was formally opened January 15. Dr. J. H. McCulloch and Dr. W. U. Tieche, who operate the hospital, were hosts for the evening. The building is one of the finest in the State.

The dates for the annual meeting of the West Virginia State Medical Association have been announced for May 19-21 at Clarksburg. The Stonewall Jackson Hotel will be headquarters.

Deaths

Dr. Hugh Payne Hirst, Leetown, aged 71, died December 20 in Shepherdstown.

Dr. Jefferson T. Hartly, Liverpool, aged 78, died November 11 of heart disease.

Dr. Adam T. Mairs, Charleston, aged 74, died January 10 from heart disease.

Dr. Walter M. Dalbey, Wheeling, aged 56, died in San Bernardino, California, January 9.

Dr. E. N. Miller, Huntington, aged 59, died February 13.

Dr. F. D. Marcum, Wayne County, aged 55, died February 13.